ROYAL COMMISSION ON MATTERS OF HEALTH AND SAFETY

ARISING FROM THE USE OF ASBESTOS IN ONTARIO

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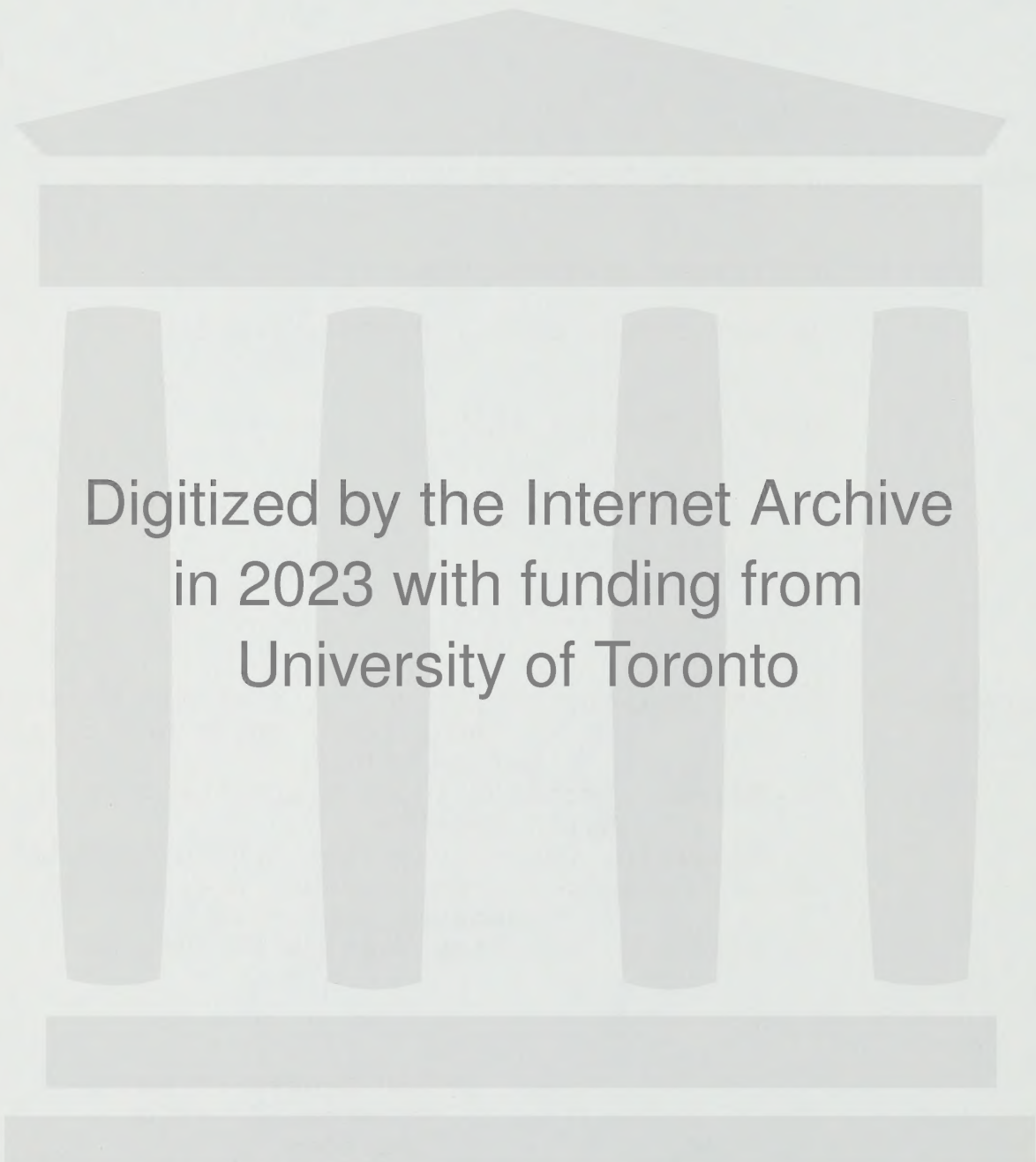
180 Dundas Street
Toronto, Ontario
Thursday,
June 25, 1981

VOLUME XIII



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THE FURTHER PROCEEDINGS IN THIS INQUIRY RESUMED PURSUANT TO ADJOURNMENT

APPEARANCES AS HERETOFORE NOTED

DR. DUPRE: Good morning, class. Mr. Laskin has been detained and urged us to go ahead without him, as does Mr. McNamee, so Mr. Nelson, do you wish to continue?

MR. NELSON: Yes. Thank you very much.

DR. JOHN CORBETT McDONALD, PREVIOUSLY SWORN, RESUMES THE STAND

CROSS-EXAMINATION BY DR. MARK NELSON, CONTINUED

Q. I guess I would like to begin asking some questions, continuing from the paper in tab eighteen on exposure data. I would like to refer to page 13 of that paper, Dr. McDonald, in which you say that, there was area sampling, monitoring, for asbestos done...four thousand samples, approximately, over seventeen years, 1949 to 1966.

A. Right.

Q. That's an average of less than one test a day, when I divide seventeen...four thousand samples by seventeen years, I got two hundred and thirty-five samples a year, less than one a day. And this was to look at exposure for over five thousand job

Q. (cont'd.) classifications, five thousand, seven hundred, eighty-three, to be specific, and for a cohort of over eleven thousand workers.

My first question is, do you feel that this kind of sampling is able to give, in terms of how limited the sampling was and that it was area sampling, not personal sampling, how accurate can the estimates be of the exposure of the individual workers during this time period?

A. How accurate is very difficult to say. First of all, I think that perhaps, to be more precise, it would be useful to look at what the sort of pattern of frequency was, because it certainly wasn't a matter of sampling, shall we say, every other day, or anything like that. That wasn't the type of sampling this was.

It consisted essentially in defining within the various mills...and I can't remember offhand how many mills there are...let us say ten, about that, I suppose...identifying in each mill a number of sites. Again, I can't remember how many in each mill would be chosen, but let us say it was...I don't know, shall we say twenty to fifty, or something like that, and then the pattern of sampling was essentially annual.

So this concept of per day is a misleading concept. It's really a matter of a number of places chosen by Mr. Lachance, the hygienist, as being, as best he could, representative of the area, and then taking a measurement at roughly annual intervals. There were some departures from that, I think. There might be occasionally more, and so on.

So that what you have at the end of the day is a pattern of...so that, if you like, for the period it concerned, which is from about 1950, I've forgotten...

Q. 1949 to 1966.

A. 1949 onwards, you have a pattern over that period of years which doesn't give you too bad a picture of what

A. (cont'd.) you might call the general level in that area.

Now that area would be quite a big area. Mills are things with floors, and the system is that in general the ore starts at the top and works down through crushing machines and separaters, and eventually it gets, the refined asbestos gets bagged at the bottom.

Again I can't remember, but let us say that on a given floor there mightn't be more than half a dozen samples. Now, a floor might be certainly as big as the floor of this building, if not bigger. So one must be very modest in claiming that it represents anything like a precise picture of environmental conditions, even if they had been done daily.

On the other hand, the pattern of concentrations, particularly when examined as they were by Mr. Gibbs, Dr. Gibbs, does give, I think, a reasonable picture of what the prevailing level of dust in those floors was.

The bigger problem...I'm not so bothered, if you like, about that as a picture of the general area conditions. A much bigger problem, two bigger problems, are that of course it did only start in 1949, and anything before that...there were the odd counts, but that's all there were and therefore anything before that is much more dubious.

The procedure, and Dr. Gibbs will be here later, I think, in this hearing and he should perhaps be questioned directly on this...the procedure in general was to try to use again all the information available on mechanical changes, control changes, ventilation, etc., information from workers, to try to put together whether or not before 1950 conditions were better or worse. In general, they were probably worse during the war years, perhaps a little better before the war years.

So a very important part of the exposure, because it was at a high level, was at a time when our measurements were,

A. (cont'd.) if you like, least good.

The other point which we have to be more cautious about is the fact that a prevailing level does not...is very hard to assess what in fact was the exposure of a man, because in the mills there were things called cabins, where the workers would often go and then come out from time to time, and whether the conditions were worse or better in those cabins - probably better - again, there was a lot of variation.

So, you know, I will share any doubts about these estimates. All we can say is that they were an honest and a skilled attempt to estimate prevailing conditions.

Q. If, as you say, area sampling are not as good as sampling a worker, as a personnel monitor, for estimating the exposure of individuals, in that these were area samples and not personnel samples, and if, as you say, at times workers would be working in cabins where the exposures might be lower, couldn't that lead one to conclude that the risk estimates were too low, and that the estimates you got in area sampling are higher for asbestos exposure than the workers were actually exposed to, due to the fact they were in the cabins during their work or during certain periods of their work?

A. The estimates that were made by Dr. Gibbs were based upon taking that into account.

Q. How could he take it into account if he didn't measure...if there were area samples, but they weren't in the cabins?

A. No, no. I'm sorry. I didn't make myself clear. In estimating the exposure of a man in terms of to what environmental level was he exposed, his movements on a floor were studied. In other words, the estimates were made of roughly what portion of the time would man spend in the cabin or outside it.

The...I think the question of the personal sampling

A. (cont'd.) is a bit of a red herring, because all attempts or all efforts at controlling work conditions are based upon area sampling. They are not based upon personal sampling. There are no data relating to the health of workers, as far as I know, of any industry, based upon personal sampling.

Personal sampling is a very useful thing if you want to know, if you want to study, let us say, what does an area level of exposure mean in terms of personal exposure. There is quite a lot of study of this, and indeed I think the future and more sophisticated methods of environmental control will depend on detailed studies of the relationship between the two, but we have to bear in mind that the exposure which a man has is still reflected in the area of concentration in which he is working, and that has been the practice in all epidemiological studies in occupational health and safety.

Q. I think it's true that it's reflected, but one bases a standard, hopefully, on the dose-response curves of exposure and disease, and it would seem to me that the most accurate way to get a picture of an individual's exposure is by sampling - having a sampling machine on that individual, as opposed to an area, and that if one is talking about those dose-response curves one would have to say that those samples...

A. Okay, you are obviously talking about...excuse me...the future.

Q. ...were more accurate.

Well, I'm talking about the present and the past.

A. Well, I mean that means the future. Because I mean, in other words, from now on we might be better to incorporate more personal sampling so that in fifty years time we will be able to assess the risk associated with personal sampling, but that is the situation. You see, we don't have it and therefore at the moment we have got to use the available data

A. (cont'd.) on environmental levels, again, with all their faults.

This is one of the few studies in the world which has any data upon exposure, so that, you know, we have to say right, are we going to just reject it or are we going to say it is the best estimate that exists at this time?

If you think the estimate is too high or too low, then one would want to know on what basis you think that, and I could discuss it.

Q. One of the other issues you've raised, as you mentioned this morning, is that few measurements, if any, existed prior to 1949.

A. Right.

Q. One of the questions I have is, can you tell us how many person years of exposure occurred prior to 1949?

A. A lot. I couldn't tell you. A lot.

Q. Would we have any idea in terms of that number, it's approximate size or percent, in terms of occurring before 1949?

A. That is an answerable question, but I couldn't answer it without a lot of study.

Q. I guess one of the corollary questions to that would be, what percent of person years of exposure that you categorize as low exposure, for which you said there was...let's see, there was no lung cancer found that you thought was statistically significant, what percent of that exposure would have occurred before 1949?

A. About the same. You know what I mean? The relative proportions, the distribution of years of work in this cohort, whether they were heavily exposed or lightly exposed, would cover essentially the same year, time period. Essentially the same.

This cohort..I mean, what is it...born 1891 to 1920,

A. (cont'd.) so it...I think the youngest employee we had in that was a boy employed at twelve years of age.

So you could say that employment would have started about...employment experience of this cohort, started in around 19...shall we say 05. Now that, of course, is taking the limit. Probably the important employment history of these men started from about 1915, 1920, so in answer to your question, you can see that quite a lot of work exposure in the cohort as a whole would have been before 1950.

Q. Okay. As you said, we don't have really good data on those exposures.

A. But we have some, yes.

Q. My next questions refer to the question of conversion from measuring dust, to fibers, with regard to asbestos exposure.

I would like to know if you feel if the dust counts or fibers counts, which of those methods provide a reflection that's most accurate, of asbestos exposure?

A. You mean a direct fiber count or a direct dust count? Or dust counted in a conversion?

Q. A direct fiber count...

A. Or a direct...

Q. ...as opposed to a direct dust count?

A. I think...I'm not an expert on this, but I believe that the variations in fiber count are worse than in dust counts. I think we can get more repeatable measurements with the dust counts, because the fiber count mechanism is not so easy to standardize.

But one is measuring fiber. I mean, like, which is giving you the best estimate of fiber concentration? Certainly the direct fiber count.

Q. One of our concerns, of course, is that in an area such as a mine or a mill there is likely to be a lot of

Q. (cont'd.) material in the air which is not asbestos, and which might, with the dust count might very well be picked up...

A. Right.

Q. ...and it could be regarded...of course, in extrapolations to a dose-response curve when it really wouldn't accurately reflect exposure?

A. Yes.

Q. We are impressed by the fact that the NIOSH/OSHA asbestos work group, they...in 1980 they recommend to look at fibers as opposed to dust, with the newer technique, the membrane filter technique?

A. I think that's entirely reasonable.

I think though, if I may comment here, that there is here...I mean I agree with you that on the face of it you would expect a higher ratio of dust to fiber in a mill than in a processing plant. Is that what you are saying?

Q. Yes.

A. Yes. And that's one of the reasons why we have been looking with Dr. Dement at this plant in South Carolina, where the textile plant with the apparently considerably higher dose-response risk, because what is rather peculiar there...and none of us can really understand it...and that is that the conversion is of the same order in that plant as in the mines and mills. We would have expected a difference.

DR. UFFEN: When you say of the same order, do you mean...?

THE WITNESS: Around three. The conversions that Dement used were of the order of three to eight.

DR. UFFEN: Frequently people use order of magnitude...

THE WITNESS: We are using about three. So in other words, we are using in the mines and mills a conversion

THE WITNESS: (cont'd.) that is very close to the one which has been employed in the textile plant, and that makes one wonder a little bit.

DR. NELSON: Q. I was looking at this 1974 paper by Gibbs, which will be in the record in the future, I'm told, and which you footnote on page 71 of your article. In that paper he says that at that time the conversion, in his mind, does not seem possible.

I would like to know if...

THE WITNESS: A. Is not impossible?

Q. He says it does not seem possible, in his 1974 paper.

A. Yes.

Q. I would like to ask you if you feel methods have improved significantly in this area?

A. No, I don't think they have. I don't...I mean, I think...I don't necessarily agree with 'do not seem possible'. I mean, it's a matter of how possible and how...you know, what degree of error, etc. But I agree with what he is saying that in fact we've only got to look at the enormous range of conversions to see how a single conversion factor is clearly a very crude averaging process.

But the methods of measurement of dust and of fiber are the same now as they were then.

Q. Okay. Yeah, I think his point was that he saw no correlation that he could understand or make sense of, between the measurements of dust and fibers?

A. Yes. I did mention yesterday that I think that is very much the reaction of a physical scientist who is not very impressed with a correlation of around point four.

DR. UFFEN: Impressed, but not favourably.

THE WITNESS: Exactly. But when we look at the correlation between dust exposure and radiological change in

A. (cont'd.) asbestos workers, correlation tends to be about point two five.

In other words, in biological problems we just can't get that kind of correlation that physical scientists like to see.

DR. NELSON: Q. On the third paragraph of your abstract you talk about averaging lower dust concentrations at six point six million particles per cubic foot, and convert that out to twenty fibers per c.c. Now, the conversion factor you are using there is around three, I believe.

THE WITNESS: A. Which paper are we now talking about?

Q. I'm sorry. It's the same one, eighteen.

A. Yes, okay.

Q. That's the paper I'll be referring to.

A. Page?

Q. Well, in the third paragraph of the abstract.

A. Mmm-hmm.

Q. You convert the lower dust concentration of six point six million particles per cubic foot, in that paragraph, you converted, I see here, to twenty fibers.

A. Oh, yes, yes.

Q. My question is, how did you arrive at a conversion factor of three? Why didn't you use, for example, a factor of...one of the other factors such as one, which you mentioned on page 21 of this article, or a factor of point three, which you mention in another article, in that there is a very wide range - over one hundred times - in terms of the conversion estimates one can choose.

A. Yes.

Q. It seems to me that the estimates of fibers would be very different if you used a lower number than three.

A. Or a higher number.

Q. Yes.

A. I mean, we could have chosen point three or, shall we say, twenty.

We chose the average there purely...this is not... the sentence starts 'if'. As it happens, I mean if you read the article throughout, we are really saying that at this stage we certainly were not serving up the results in any way in terms of fibers.

It so happens that this six point six million particles, or about twenty fibers, does agree fairly well with, in fact, what our estimate eventually was, of an average of three point one four. So I suppose we were really quite close to the average.

I think choosing the average is not an unreasonable thing to do.

Q. You say later in your article, on page 21, that a study of this kind is as good as the environmental and mortality records on which it is based. In light of the fact that there are serious limitations on the accuracy of the exposure data...things we discussed this morning...that their area samples, that only four thousand were done and few, if any, measurements were made prior to 1949, that there are serious conversion problems and a wide range of conversion factors. Would you say that the conclusions you reach in that third paragraph with regard to finding no statistically significant lung cancer at lower exposures...which are estimated to be twenty fibers...would you say one can really say that with a high degree of confidence?

A. How high do you want me to say it?

Q. How confident would you regard that statement?

A. I can't really ...it's my best estimate.

Q. I guess the reason we feel that's important is it seems that some people have inferred a lot from that last paragraph, namely that there is no significant lung cancer at

Q. (cont'd.) twenty fibers per c.c.

A. Yes.

Q. which is regarded as a low exposure, and we feel in light of the limitations of exposure data and no real measurements from 1949 or before, or very few measurements, we are just wondering with what confidence one can say that there is no lung cancer at that level.

A. As a matter of fact, I think with a fair amount. I say this because there is...I can assure you that I doubt whether there, anybody in this room is as conscious of the problems of estimates as we are, but one of the interesting things is that with this enormous problem of estimate, and variation, that exposure-response line for lung cancer is one of the most precise estimates of exposure response in cancer published in any field today.

It's worth noting. I think there are only two other areas which are perhaps more precise than this, and one is in radiation workers and the other is in relation to cigarette smoking.

The point is that error leads to variation. If our error was great, we would not expect our points to fall so close to a line.

Now, that is a form of internal consistency.

Now, what would be perfectly fair..in other words, what that implies to me is that on average our estimates are quite good, but what we don't know...I did speak about this yesterday...is, we do know that there is random variation on measurement, and we do know equally, as I was describing yesterday, that the effect of random variation in measurement is to reduce somewhat the slope of the line. We estimate very roughly that that line, in the light of unpublished current information, the slope of that line may be depressed by about sixteen percent on account of variation.

A. (cont'd.) So I suppose that's the kind of reaction I have to this issue. I think that it is an estimate that has a very high degree of probable truth. The exact slope may vary a little bit.

Q. Let's assume that it is depressed sixteen percent.

A. Yes.

Q. Would that change what you find, in terms of lung cancer being statistically significant?

A. I doubt it.

Q. Well, I'm..

A. It would simply give you a different slope line. It wouldn't affect the relativities, you see.

DR. MUSTARD: Can I ask a question about the statistical significance?

In your responses here, if you had three million people your argument about the statistical significance would change?

THE WITNESS: Completely, yes.

DR. MUSTARD: So that indeed it's statistics, as I understand, that you are arguing from a cohort that you have studied?

THE WITNESS: Exactly.

DR. MUSTARD: If you increase the numbers, indeed it might be statistically significant?

THE WITNESS: It certainly would be. Any difference becomes significant if you have enough people.

DR. NELSON: Q. I think you've already said that we don't know what percent of person-year exposures occurred prior to 1949, but that this could be significant.

You said on page 21 of your article that for the more important earlier years we have only estimates to go on, and I'm quoting you.

A. Right.

Q. And yet you maintain that you have a lot of confidence in this estimate. I fail to understand that?

A. That's fair enough. It's a matter of opinion.

Q. I think it is a matter of opinion. Yes, I agree with you.

A. I've studied it for many years, and I've come to the conclusion that it looks true to me, but of course I might be quite wrong.

Q. I think we would agree that the size of the study population, or the manner in which it is divided, can influence the degree of statistical significance we find or don't find.

A. Yes.

Q. If we look at table seven D, of this paper, tab eighteen, which is located on page 18, this is estimates of increasing...we are looking at mortality here for gross service twenty or more years, and we find that the population of workers here has been broken down in various columns according to estimates of their exposure - low, medium and high.

A. Mmm-hmm.

Q. Again, I'm referring back to the last paragraph of your abstract on the first page of this article in which you say, "No statistically significant lung cancer was found".

But in fact even within the columns, when one looks at the SMR's, standard mortality ratios, even for the low exposure, for lung cancer it's one point two one, and for esophagus and stomach it's one point three six. Would you say those SMR's are significant?

A. I don't know. I would like you to note, I mean before you start drawing conclusions, that how would you then take the SMR for esophagus and stomach, one point three six for low exposure, and point six four for medium exposure? You would need

A. (cont'd.) to take them both together, wouldn't you?

Q. When you say lower dust concentrations I was assuming you meant low exposure.

A. I meant that.

Q. You mean low and medium?

A. Well, in point of fact, as a member of the Commission has already said, I think we mean those two groups together, but I think what you see there is, if you like, part of the variation. It is not reasonable to believe that people with lower exposure have a higher risk of esophageal and stomach cancer than those with medium exposure.

Q. I agree with that. I think the point we make is in light of the problems with measuring exposure accurately, this could be a reflection of inaccurate exposure measurements.

A. Yes. It could be all sorts of things, you know. I do want to emphasize that.

We are not dealing with, in studies of this kind we are not dealing with either human experiments or experiments with experimental animals. That's one of the big problems of interpreting epidemiological data. We are not dealing with experiments. We are dealing with human beings and the way in which they move.

You could say that there is no absolute validity in any epidemiological study whatever - none. And I would support that, but in the life in which we live we have to put together the information as best we can, and inspite of the fact that we know that our information on, shall we say lung cancer and smoking, is not valid, it has no more validity than this kind of stuff, nevertheless society finds it useful to consider what information there is on smoking histories and lung cancer.

I emphasize this because we are not dealing

A. (cont'd.) with laboratory experiments on human beings. People who are of light exposure are not necessarily the same as those with heavy exposure. We cannot look at precision. We have to look at patterns and decide whether they seem reasonable or not reasonable to us.

Q. I agree with you that many of the questions relate to how one interprets the data, and clearly we have a difference of opinion on how it should be interpreted.

With lung cancer, for instance...

A. I'm not sure that we do. You know that, I don't.

Q. Well, it seems to me we do. If you look at the low and medium exposures for lung cancer, the SMR for lung and the low is one point two one, and for the medium is one point 0 eight. If we average those we get one point one five, which means a fifteen percent increase in lung cancer for people exposed at these concentrations. Do you think that's an important statistic?

A. Could you repeat the question?

Q. Well, you said we would have to take into account the SMR's in the low and medium groups...

A. Can I cut this short, because I'm not sure what we are trying to get at, you see. It seems to me that what you are trying to work towards is some justification of a statement that there isn't a risk below a certain level. I have emphasized again and again that that is not what we said.

I mean, if you are trying to prove there is no risk below twenty fibers, then I can go on in this and discuss it with you. But I'm not trying to prove that, so you know, is that the gist of your discussion? I'm not clear about it.

Q. No. The gist of my discussion is that in the third paragraph of the abstract you say that there is no excess mortality that could be considered statistically significant, except for pneumoconiosis, at twenty fibers.

A. That's right. Do you think that's untrue?

Q. Well, I think it's a question of what one looks at at the table. Certainly an SMR at a low exposure of one point two one for lung cancer, that's a percent increase of twenty-one percent. It seems to me that's significant.

A. Well, I don't know. I mean, I'm taking judgement of Professor Liddel, a professor of medical statistics at McGill University, that he thinks that's not statistically significant.

Now, I mean, you know, it's a matter of fact. If he's wrong in his calculation, I think that should be demonstrated. Nobody has demonstrated that. I'm inclined to believe it.

But I still argue that that's irrelevant, because the purpose is not whether it is significant or not. What we are saying is that due to the inability of studies to detect certain increases or decreases in mortality, we have to fall back on the pattern of exposure response, and our best available estimate of what is the risk at any level below twenty fibers will be based on that. And that's what this last paragraph said.

DR. MUSTARD: Can I ask a question again here? Because I wonder if we are not debating something which is just statistical.

You have identified that you have...there's error in measurement, there's error in estimates of what's happening to people.

THE WITNESS: Right.

DR. MUSTARD: Those areas are part of the variation which affects statistical probability, is that not correct? Am I right? Those errors that affect your statistical analysis?

In other words, if you have a fair amount of error in your estimates, and small numbers, showing a statistical probability is more difficult than if you had a very precise

DR. MUSTARD: (cont'd.) measurement?

THE WITNESS: Yes. This term 'error' didn't need to be examined. What we mean statistically by error, is variation. Error implies that it is incorrect.

DR. MUSTARD: I mean variation.

THE WITNESS: Variation, right.

Yes, it is a reflection of variation. If we have true error in the sense of bias, no statistical significance can take care of it.

DR. MUSTARD: But in the sense that the variation had been very small for the things that went into the calculation for those nineteen hundred and four men, that might be read a little bit differently? It might be statistically significant, isn't that the point that you are trying to make, is that the uncertainties in here create the problem...or if you had two million men, it might be slightly different in terms of calculation?

THE WITNESS: It's mainly the latter. This, I am virtually sure, is a test of significance of the kind that we have always tried to avoid using, which is taking data at face value. It is not taking any account of variation.

If we took account of variation, I don't suppose any of the paper would be statistically significant. Or any other epidemiological paper.

DR. NELSON: Q. Let me try to understand your feelings on this. Would you say that based on your third paragraph in the abstract that at twenty fibers or less, if you regard as low exposure, that lung cancer as a result of asbestos exposure...

THE WITNESS: A. Excuse me. I don't regard it as low exposure. What made you think that?

Q. You said lower dust concentrations.

A. Lower, yes.

Q. Okay. At lower dust concentrations of twenty fibers per c.c., would you say on the basis of the paper that workers would not have lung cancer as a result of those exposures, or that they may and we just haven't been able to detect it?

A. I don't know whether you've read the paper, but what it says is that they will have. That there is...that the extrapolation of the linear relationship implies that they will have a risk of lung cancer, right down to point zero, zero, zero, zero, one fibers per annum.

You know, that's what the implications are, that there is a finite risk with every fiber you are exposed to, including that which is naturally in the air.

DR. NELSON: We would like to hand out some material to be entered into the record...some pages from the submission to the Royal Commission on Matters of Health and Safety. I believe it was entered by the Quebec Asbestos Mining Association, and as I said yesterday, I had assumed that everybody had a copy but for those people who don't, we would like to hand them out now.

DR. NELSON: Q. I would like to refer to page nineteen of this document, to the last paragraph on that page, Dr. McDonald, in which the Quebec Asbestos Mining Association argues on the basis of your paper that it's almost impossible to discern increase in risk at an exposure rate of twenty fibers per c.c.

Do you agree with that statement?

THE WITNESS: A. That's what we have just been talking about, and that is what is stated in the third paragraph of that abstract.

Q. You, yourself, state that there is an increased risk that is significant, due to pneumoconiosis.

A. Yes.

Q. They fail to mention that here, don't they?

A. I don't know.

Q. Well, I don't see it, do you?

A. I didn't look. I'm looking at one paragraph. I don't see the word pneumoconiosis in that paragraph, no.

Q. I don't either.

A. No. I don't know whether they are referring to pneumoconiosis or not, because that may be mentioned earlier.

Q. Well, on the second paragraph there, under Findings, they talk about the relationship, dose-response relationship in cases of asbestosis and lung cancer.

A. Oh, yes.

Q. I just think it's important that when industry makes a statement like that which is so blatantly inaccurate, that that needs to be pointed out.

I would wonder...

MR. CASGRAIN: Mr. Chairman, I think this was a purely gratuitous assertion which should not be on the record. This is not the time to do this. If my learned friend wants to argue in due course, fine, but I can't stand here and accept that statement as attorney for QAMA, and I don't.

DR. NELSON: Well, workers' lives are at stake...

MR. CASGRAIN: And if we want to embark now on a discussion, I will. But I don't think this is the time.

I would ask my friend to perhaps refrain from that argument.

THE WITNESS: Mr. Chairman, I would also like to be sure that I am not in a position of having to comment on somebody else's document.

DR. NELSON: Well, I think what we are talking about here is, we are talking about a standard for asbestos...

THE WITNESS: A. Yes.

DR. NELSON: Q. ...and statements are made in

DR. NELSON: Q. (cont'd._ this document that have been submitted to this Commission, and I would like to know your comments as a scientist on the validity or invalidity of these assertions. I think that's a very important part of these proceedings.

THE WITNESS: A. I think if you think this document is important, I suppose I should have had it to read before.

Q. I'm not responsible for distributing the documents.

MR. CASGRAIN: Now everybody is guilty.

MR. LASKIN: Well, Mr. Chairman, just two points. Number one, my friend is here to ask questions, I think, not to express his own opinions. If he wants to express his own opinions he can surely get in the witness box and we'll have a chance to question him.

Insofar as Dr. McDonald is concerned, it seems to me we have discussed this third paragraph in the abstract now for about forty-five minutes, and I don't know what we are going to add to it by asking Dr. McDonald to give his view on what somebody else said about his writing. He has given his own view directly on what he said his article means, and it seems to me we should all be content with it. I think we all understand what Dr. McDonald said.

DR. NELSON: Well, maybe you understand it, but we don't, and I don't appreciate attempts to censor our questions in this area. If you want the participation of working people in the OFL in these hearings, we intend to participate. If you don't, then you should make that clear. It is not at all clear to me, you know, what is being said in Dr. McDonald's paper and comments regarding it.

So let us decide what is clear and what is not, insofar as we are concerned. Thank you.

DR. DUPRE: I think the chairman should now come

DR. DUPRE: (cont'd.) to the aid of parties, if it could try to do so. First of all, I think we should all try to remember that we are here today, as we are on any day when we have a scheduled witness, to try to get the maximum amount of help from that witness that we can.

Secondly, I certainly would not want any party to feel that he should be unduly confined in posing questions, but at the same time I think all parties should be sensitive to what I would call gratuitous editorializing.

As for Mr. Laskin's point, I don't know about my colleagues, but I have a certain sense of deja vu in terms of the points relating to the third paragraph of that abstract, although admittedly this one has been raised in a brief that was put before the Commission...a brief which, by the way, I might point out in a reminiscent mood, I remember. Of course, I have read all of the briefs and I think the record of the meeting will show that I complimented the QAMA for all the information they gave us in their brief, which as I put it, certainly stated their interests in a straightforward fashion...the same compliment as I gave to the OFL on their brief when they submitted the day before. It was indeed, again, a very comprehensive document that I felt represented the interests of the party well.

Now, at this point one reason why we have guest witnesses who formally testify is that although you can take it for granted that the Commissioners are not necessarily very bright, on the other hand simple physical examination will bring home to you that none of us was born yesterday. And indeed the whole purpose of this kind of testimony is to enable us to understand in context briefs from organizations, which we know offer a very useful compendium of information which, of course, is mixed in with the usual amount of slanting and/or BS and so on, but that is part and parcel of these kinds of

DR. DUPRE: (cont'd.) exercises. So that's why we have these different phases in our hearings, and of course what goes on in testimony, in many ways, is something to balance what we all know are honestly-meant, honestly-presented presentations from parties who have an opportunity to speak to their interests.

So may we now take it that in these pages from the QAMA brief you are bringing to our attention that Dr. McDonald's testimony certainly would indicate that what is stated in the QAMA brief should be read with considerable care?

DR. NELSON: Yes, thank you.

THE WITNESS: Mr. Chairman, may I make a statement?

DR. DUPRE: Please, Dr. McDonald.

THE WITNESS: I feel a little unhappy that, in a sense, the implication has been that I have in any way either failed to promote the information of our work to the workers who are concerned for their understanding, or that I have promoted the misunderstanding part of our findings.

I would like to emphasize that for at least ten years our publications have promoted increasingly the view of a direct relationship between asbestos exposure and the probability of cancer...longer than any other group of workers. We have done this in published paper, in public hearings, in meetings with trade unions concerned, and I do resent a little the suggestion that we are in any way trying to mislead anybody.

DR. NELSON: Let me say that the intent of my questions was not to imply that you have misled. The intent of my question was to raise a very important statement, we feel, that is based upon your paper, on pages nineteen and thirty-two of this document submitted by the Quebec Asbestos Mining Association. Page 32, they repeat the statement that it has

DR. NELSON: (cont'd.) been difficult to establish an increasing risk at twenty fibers, and again we think it's important, in light of your paper, to point out that risk has been established at least for lung fibrosis due to asbestosis, and that this is a serious omission...or one that people should consider, the lack of discussion of pneumoconiosis.

In that line, may I ask you, Dr. McDonald, a question about pneumoconiosis?

I would like to refer to table two on page 15, where the number of deaths that were attributed to pneumoconiosis, which I believe yesterday...that's page fifteen, table two, tab eighteen ...

THE WITNESS: A. Yes.

DR. NELSON: Q. I believe you said yesterday most cases of asbestosis were classified as pneumoconiosis. I was wondering if you could comment on if you feel it's possible that lung fibrosis which resulted from asbestosis may have influenced or played a part in some of the other deaths listed here, such as heart disease...of which there were over fifteen hundred...such as respiratory tuberculosis, and other respiratory diseases?

THE WITNESS: A. Yes, I do think it possible. I, indeed, think it likely.

To put a quantity of contribution to those is hard, but yes, I do for two reasons...three reasons.

One, because in heavily-exposed men I'm looking for the evidence. I think perhaps some of the evidence...I don't want to go into detail, but probably some of the evidence would be found in page eighteen, seven D table, where I expect we'll find...which we do find...say, on heart disease, evidence of a gradient in relation to exposure. All right? It isn't ever so dramatic. It goes up from an SMR of point eight seven, point nine five, one point 0 six, one point one two. So there

A. (cont'd.) we have a gradient which is compatible with that hypothesis that the dust disease...because there is no mechanism that I can think of of asbestos attacking the heart direct, it seems likely that this is an indirect effect by putting an extra load on the heart from the degree of damage to the lungs.

Okay. I think in fact most causes of death are...show some increase in the high-exposure group, and I think the reason for that is another sort of truism, and that is that you die from two causes more easily than from one. You know, death is a result of a number of disease processes, and the more you have in your body, the more you are likely to succumb from any one of them.

So, yes, I think we must assume that any debilitating disease, if it gets to the point of putting an extra load on your survival chance, will not necessarily kill you from that point, but may increase your chance of dying from something else, yes.

Q. Thank you.

If I could just have one minute?

Okay, we would like to enter another item into the record, which is from Sam Epstein's book, The Politics of Cancer. It's a quote that he takes from a document we would like to distribute now.

MR. LASKIN: I wonder if Mr. Nelson formally wants these documents in the record? Do you want them marked as exhibits?

DR. NELSON: Yes, Yes.

MR. LASKIN: I suppose we should if we refer to them. I suppose the extract of the QAMA brief would be whatever exhibit number we are now up to. I defer to the back row here.

MR. WARREN: I would guess twenty.

MR. LASKIN: Twenty?

MISS KAHN: Yes.

MR. LASKIN: Let's make this extract from the Politics of Cancer, exhibit twenty-one.

EXHIBIT # 20: The abovementioned extract was then produced and marked.

EXHIBIT # 21: The abovementioned excerpt was then produced and marked.

DR. NELSON: Q. what I would like to refer to is on...the first page of this is just a copy of the title page. The second page, the last full paragraph there is a quote that Dr. Epstein has taken from a Quebec Asbestos Mining Association pamphlet, in which they are talking about the risks of asbestos and they say, "Can a little bit of asbestos kill you?

The response is, no. Long-term medical studies of workers who were exposed to asbestos show that low-to-moderate levels of exposure do not lead to an increased rate of disease. In these studies, the higher-than-normal incidence of disease was found only among employees exposed to extremely high asbestos concentrations for long periods of time".

I would like to know if you think that statement is an accurate reflection of the risk that workers face due to asbestos exposure...

MR. CASGRAIN: Mr. Chairman, I object again to this sort of thing. What are you trying to do here? I have read the page in question...my learned friend is trying to do what now? If he wants to argue with me on QAMA, I'll go anywhere with him and argue.

DR. NELSON: I'm not arguing.

MR. CASGRAIN: At this level here I don't see why we should involve Dr. McDonald in it...

DR. NELSON: I'm not arguing about QAMA. I would like his opinion upon a statement made with regard to the risk of asbestos exposure, which relates to the setting of a standard. A statement is made here, a scientific statement that there is no risk of disease at low levels, that disease is only found at high levels, and I would like his comment on that assertion.

DR. DUPRE: Dr. McDonald, I take it that what Mr. Nelson is asking you, and I will permit the question to the extent that I understand it's impact, he is asking you - in your expert capacity, whether in your opinion the statement that is quoted here from a party that also has an interest, is a statement that is supportable on the basis of the studies in which you are knowledgeable.

I will, Mr. Casgrain, with respect, I will permit the question in that sense.

THE WITNESS: A. It remains a difficult question, a little bit like when did you give up beating your wife, or whatever.

But...because in sense they are both wrong and they are both right. Can a little bit of asbestos kill you? Yes, of course it can. We know that. So the answer then is, no. So that's wrong. A little bit of asbestos can kill you.

"Long-term medical studies of workers who were exposed to asbestos show that low-to-moderate levels of exposure do not lead to an increased rate of disease..." of disease...low-to-moderate levels of exposure...I don't quite know where that...I'm sorry. I'm sure you want me to be careful over the words, so low-to-moderate - he doesn't assign what he means by low-to-moderate, because the answer to that will obviously be

A. (cont'd.) what we have been discussing for the last hour, that there will be a relationship directly related to how much, okay?

"In these studies, a higher-than-normal incidence of disease was found only among employees exposed to extremely high asbestos concentrations for long periods of time".

Well, that's not true because we certainly know that there have been studies which have shown a high incidence of asbestos disease after very short periods of time, for example the gas-mask workers.

On the other hand, we have got the general fact that there is this increase and we have again been arguing about the fact that in, say the mining and milling study, and I presume that's probably the context of the OAMA statement, that it is difficult to detect disease below what in fact is low or moderate.

Now, as I said, the difficulty to detect is not the same thing as the absence of. So again, you see, it isn't... I'm trying to look at the thing on all sides. What is true, of course, is that there is a relationship between the clearly asbestos, specific asbestos diseases and dose, namely lung cancer and asbestosis. To a lesser degree with the diseases which are clearly indirectly related...I mean such as respiratory disease, cardiorespiratory disease, and then some evidence that almost any kind of disease will be increased in relation to asbestos.

So, you know, I'm picking several points about this statement which are untrue, but on other hand, of course, I am pointing out there's elements of truth in it.

I'm sorry I can't do better.

Q. That's very good. Thank you.

MR. CASGRAIN: Mr. Chairman, if I may make a

MR. CASGRAIN: (cont'd.) statement at this stage?

DR. DUPRE: Yes, M. Casgrain.

MR. CASGRAIN: We now have the strange situation where a question has been asked of a witness, which is nothing to do with the statement made, of a statement which would have been made by my client and to date which is not pointed out, and which is reported to have been made by my client by somebody I don't know, called Mr. Epstein, that I have never seen.

Now, I just want to point out to you that this is obviously the weakest kind of information you can ever put to a witness. This is number one.

I suspect that this article to which Mr. Epstein referred may have been quoted properly, but I don't know about that and neither do you, because we have no evidence in this respect.

I suspect as well that the date of the article may have been 1970, but we don't know, do we? I think it's probably 1970. I can't confirm it.

Having said this, it shows to you that it may be that in 1970 the information behind this article was not as complete as it is today, and we could go on arguing forever that NIOSH in 1972 didn't have information sufficient to permit it to go as low as it went down to.

So when one asked the witness to tell you whether we are wrong or right, we may be wrong today in the light of the information that we now have. Were we saying something wrong in 1970? Nobody knows that.

Now, I make this, for the record, because it would be unfair to otherwise assume if one will read the record that this is a statement by my clients...at some date, even today.

Now, I don't propose to make a big issue and say let us produce the excerpt and have whoever wrote it come

MR. CASGRAIN: (cont'd.) here and so on...which perhaps in any court of law you would have to do. But I wanted to point that out to you.

DR. DUPRE: The point is well taken, Mr. Casgrain. I would remind you, of course, that this is an inquiry under the Public Inquiries Act. It is not a court. Along with that, of course, goes the fact that I can assure you that we don't consider any parties here to be in the dock. I will simply, once again, point out to you, Mr. Casgrain, that I stated the stipulations under which I felt Mr. Nelson posed the question, and the witness indeed at this point simply responded from his expert point of view on what he thought of the paragraph they wanted to quote.

The record shows that, and I...he could have quoted from any study or anything that he would have chosen under the circumstances.

MR. CASGRAIN: The concern is to all, too. I think the witness himself would say that his own study that he carried out, he had been refining and re-examining and going further and further into his own study which he carried out prior to 1974, so obviously what you may think of the study or how you can use it, it can vary as you find the material. This is why I say the date of that publication is of importance.

DR. DUPRE: Understood.

Mr. Nelson, do you have any other lines of questioning?

DR. NELSON: I have one more question.

DR. DUPRE: One more?

MR. WARREN: Mr. Dupre, if I might just add...

DR. DUPRE: Yes, Mr. Warren?

MR. WARREN: I tried to check in this book to determine the date of the quotation which is made, and there is no reference to the quotation. So if that adds anything to the record, there is no reference and so we don't know the date one

MR. WARREN: (cont'd.) way or the other.

DR. DUPRE: Right.

MR. WARREN: Maybe you folks can find the reference and enter it into the record.

DR. NELSON: I have one more question.

MR. LASKIN: I'm sorry. Could we at least have the date of publication of the book?

DR. NELSON: Yes. Well, that's on the first page, I believe, of what I submitted to you.

MR. LASKIN: No, it's not.

DR. NELSON: It's not? Okay.

MR. WARREN: I think it would be also useful to say that the page that he is quoting from is page 84 of the book, too, which also doesn't appear on my xerox copy.

DR. NELSON: This is the 1979 Anchor Press edition, Politics of Cancer.

DR. NELSON: Q. If I could refer to one of the papers we have submitted today, the submission to the Royal Commission by the Quebec Asbestos Mining Association, Dr. McDonald, to page 20, where they have a quote from a study, I believe, or from a talk that you gave. The middle of the page, at the IARC Conference, of the Biological Effects of Asbestos, at Lyon, in 1972.

In this paragraph here on page 20, you are talking about lung cancer and asbestos...rather, lung cancer due to asbestos, and asbestosis.

You said, "It seems clear that there is a direct relationship with length and level of exposure".

Then as a part of the last sentence you say, "We conclude that the risk of a clinical manifestation of these two diseases, that it can be reduced to an acceptable level".

Q. (cont'd.) I know this issue was raised yesterday and I guess my question is, in light of the fact that you have used that term, acceptable level, in this paragraph, if I could ask you what you mean by acceptable level?

THE WITNESS: A. I didn't mean any level there. I meant that society having to find an acceptable level, I had a belief that that level could be achieved.

That would be my kind of reaction to that question.

You will also bear in mind, incidentally, that...and I think perhaps it ought to be stated here because we have had the last hour or so discussion with no particular emphasis on the fact we are discussing chrysotile mines and mills...

Q. Yes.

A. Everything that we have been saying is focussed on that, and I wouldn't want to extrapolate beyond that without a lot of thought, and certainly this particular statement, though I still don't quite recognize it, it's a bit of a problem here because J.C. and A.V. McDonald, so far as I am aware, did not make a paper in 1972.

I did, yes, so I don't know what paper this is now quoting from. I don't even know if it is a quote.

I'm sorry. I have all my papers here, so if one had a reference, I could look it up.

Q. That's quite all right. I'm sorry I don't know where it's from either. This is from the brief.

A. No, but I suppose that I would still stand by this, that I do believe that on the data that we have available by any likely level of acceptability, it should be possible in asbestos mining and milling to get an acceptable level.

I really have no serious doubt about that.

Q. Okay.

A. And that is within sort of common-sense concepts of what might be acceptable.

DR. NELSON: Thanks very much. We are through

DR. NELSON: (cont'd.) with our questions.

DR. DUPRE: Very well.

Should we consider taking...giving our witness a brief five minute respite, perhaps, and get back at twenty-five past?

THE INQUIRY RECESSED

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THE INQUIRY RESUMED

DR. DUPRE: Mr. Warren?

MR. WARREN: I think I've been elected.

DR. DUPRE: Sir, please proceed.

CROSS-EXAMINATION BY MR. WARREN

Q. Dr. McDonald, we have been talking now for more than a day about the limitations of epidemiological study. At the same time I think you have testified that epidemiology, nonetheless, has its values and in the absence of these kinds of studies, we know little or nothing? Is that a fair statement?

A. Yes.

Q. In trying to conduct an epidemiological study which can provide us with information or guidance about a dose-response relationship, I'm going to list five factors that it seems to me are important, and I want to see if you agree with those five factors.

The first would be the size of the cohort. In other words, the larger the cohort, the more likely it is that one can uncover an effect, first of all, and secondly, establish some dose-response relationship.

The second would be the completeness of the followup. In other words, the more complete the trace is, the more confidence one can have in the results of the study.

Q. (cont'd.) The third would be to have exposure estimates, and I realize there is a spectrum of how good they can be all the way from guesses at the one end of the spectrum, to perfect personal samples for every worker involved.

The fourth criterion that I would list would be the statistical techniques employed to evaluate the data.

The fifth would be other methodological issues, including something that Mr. Laskin started discussing early yesterday, and that is case controls within the cohort as a means of verifying the results.

Would you say that all five of those factors affect the question of whether or not a study provides useful information on a dose-response relationship?

A. They certainly do. All five are clearly relevant and important. I don't want to pick on it too much, I think size in itself is not a virtue. I mean adequate size to answer the question is obviously important.

I say this, I make that qualification simply because there is often a payoff between size and quality.

Q. Now, I'm going to go over with respect to your study, to these five categories in a moment, but have I left out anything important which bears on the quality or utility, usefulness, of an epidemiological study? Are my five factors pretty comprehensive, or are there things that I've left out?

A. Nothing immediately comes to mind. There may be others.

Q. Maybe some things will come up as we discuss the issue.

Now, on the issue of size, as you have said a moment ago, there must necessarily be some tradeoff between size and these other factors. Were we to have, say, an epidemiological study of a million people, it would be harder to do all of the other things that I discussed, including followup and exposure

Q. (cont'd.) estimates and so forth. Is that a fair statement?

A. Yes.

Q. Now, as epidemiological studies go, would it be fair to say that your cohort is a fairly large one?

A. Yes.

Q. Based on the years that you've spent evaluating this cohort, would you say that it is a cohort of sufficient size to give you meaningful results?

A. Yes.

Q. Now, let's go to the second factor, and that is the completeness of followup. Would you tell us what your success was in following up the members of your cohort?

A. Yes. The figure over the years has hovered around the ninety percent mark. This is not as high as one would like, and is not as high as can be achieved in countries which have social security systems which allow you to trace people.

However, we naturally have looked very carefully at this issue, and the deficiencies in tracing have been almost wholly in people of very short, relatively short exposure... employment, employment, and for the most part, in the more distant past. For obvious reasons, such people are difficult to trace by the methods that we were forced to use.

Somewhere in the paper it does say that of those people who were still alive in, I think it might be 1950, or something like that, we have in fact something like a ninety-eight or ninety-nine percent trace, and much of our data is based on that. I think we do have, if we have a reservation, it would be on the fact that the short exposures in the past are deficient and one has to consider in what direction, if any, the bias would be.

We don't know. The reasons why it may be that, it is anybody's guess, really. We have no particular reason.

A. (cont'd.) We have to consider what is the reason we couldn't trace, and for the most part it is because we didn't have a full name. You have to say is it more likely that a person for whom you have a full name will have a different cause of death than a person for whom you have the full name (sic), and we conclude it probably wouldn't make much difference.

But that is its imperfection. It certainly doesn't affect, may I say, most of our tabulations, which are based upon, as you will see, on persons who have five years or more exposure. We have lost almost nobody with that sort of duration of employment.

Q. You are familiar, I am sure, with many of the other cohort epidemiological studies which have been done for asbestos exposure. Does this followup rate stand well in comparison to those studies?

A. Yes, providing with the reservation I have made, providing that we consider that the most of the information is coming from persons with longer periods of employment.

Otherwise, I would say ours is sort of in the middle or towards the top of the league, but it isn't at the top. The British studies consistently beat us.

Q. Given the degree of followup which you've had, I take it from your testimony you nonethless feel that the followup is sufficiently good in order to allow you to draw meaningful conclusions?

A. Yes.

Q. On the question of exposure estimates, which we have talked about at great length and I don't want to belabour that issue too much, I believe you testified yesterday that the exposure estimates upon which your study is based were all done by one man or under the direction of one man?

A. Yes.

Q. Does that lend confidence, in your mind, to the exposure estimates which you have employed in your study?

A. It removes one source of variation. The confidence I have is actually by knowing the man, who is actually a very competent man. It was also, if you like, what we are really saying is that one man made virtually all the measurements.

But, of course they were judged by another man - that is Dr. Gibbs - so yes, I think that within their obvious limitations, there it is. I think we have made as good a job of it as was possible.

Q. Now, on the question of statistical techniques, you are not by profession a statistician yourself, is that correct, Dr. McDonald?

A. Correct.

Q. Over the decade, as you have analyzed your data, have you sought professional advice as to how you ought to analyze your data from a statistical standpoint?

A. Yes. I think we've always had a statistician in our group, with literally international standing. But perhaps the strongest claim we have here is that we submitted our whole study, and its whole issue of statistical analysis, to a general meeting of the Royal Statistical Society. I think ours is the only study on record where that has been done, and where this was subjected to probably the highest level of statistical, professional meeting there is, and the records exist both of the paper and the discussion on the methods. And in fact it has been agreed, I think, that our methodology has made a contribution to the analysis of cohort studies.

DR. DUPRE: Could you just permit a question here, counsel? Did I hear correctly, Dr. McDonald, that to the best of your knowledge your studies are the only studies of asbestos that have been examined by the Royal Statistical Society?

THE WITNESS: As a methodological issue, so far as I am aware, yes.

DR. DUPRE: Thank you, counsel.

MR. WARREN: Q. I take it based on that review and the scrutiny which your data and statistical methods were given by those expert statisticians at the Royal Society, you feel confident, indeed quite confident, about the methods employed and the conclusions which can be reached from a statistical standpoint in your study?

THE WITNESS: A. Everything is relative, but I think we are reasonably confident. I would like to add that in our early analyses back in 1969 and 1970, we used methods which we were unhappy about, and which we certainly would not use now. Funnily enough, they gave the same answer.

Q. I was trying to find a way to pose the question without...

THE FOREGOING HAS BEEN PREPARED
FROM THE TAPED RECORDINGS
OF THE INQUIRY PROCEEDINGS

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....to page 41

MR. WARREN: Q. The new methods, the methods which have been used since, I think it is -- and you -- maybe you can tell me ---

THE WITNESS: A. Well, certainly from 1976 onwards.

Q. --- are reflected in the compilation and evaluation of your results seen in tab 18, which we've been discussing.

A. Yes.

Q. Finally, on this question of methodology, Mr. Laskin noted, at the very beginning of his questions yesterday, the difference between cohort studies and case-control methodology.

Now, I think we all understand the cohort method, since we've had a number of witnesses already testifying regarding cohort studies. Let's leave aside for a minute the use of a case-control method within a cohort and let's talk about the case-control method separate from the cohort.

Could you explain for us all how that methodology works, and how it attempts to investigate a relationship between exposure and disease.

A. I think perhaps I can give the simple and historical important example of the first observations which were made on the association between smoking and lung cancer.

What Bradford Hill did was to pick, from a number of hospitals -- this is Dahl and Hill -- a number of hospitals, cases of lung cancer, and they picked from those same hospitals, at the same time, other patients of the same age who were not -- who did not have lung cancer.

And, essentially, what they did was to inquire from them both, by as near as possible identical methods, what were their smoking histories, and this was done by simply asking

A. (cont'd.) them. And so, what you have, then, is, if you like, a comparison of the smoking habits of lung cancer cases and the smoking habits of people who look similar -- they're in the next bed, or whatever -- and who did not have lung cancer.

Q. Now, since Bradford Hill's study on cigarette smoking, the case-control methodology has been widely employed to investigate associations between disease and environmental exposures; is that correct?

A. That's right; about the most efficient method.

Q. It's one of the cheapest methods, I think, is it fair to say?

A. It's about the cheapest, yes.

Q. And I take it, case-control methodology has been used outside of the hospital context which you're talking about; in other words, there have been general population controls used where hospital controls either weren't desirable or weren't available.

A. But I think it's worth emphasizing this point; that if you pick your cases from a hospital, you should also pick your controls from a hospital.

Q. Now, case-control methodology, as you testified yesterday, has been the subject of very considerable debate and controversy in the community of professional epidemiologists. That's a fair statement, I think, isn't it?

A. Yes.

Q. Could you state for us what some of the limitations of case-control methodology may be and what the epidemiological community has noted as possible biases or deficiencies in that methodology?

A. I think the two problems arise, first, in relation to the selection of the cases, and then in relation to

A. (cont'd.) the selection of controls.

The logic of the whole thing is that, if you take cases, a sample of cases, for study, that they should be reasonably representative of all cases that you wish to infer your findings to.

You see, if you had -- I can again give you an example of the observation that, in a town in England -- that a high proportion of patients with nasal cancer had worked in the manufacture of wooden chairs. The man in question selected his cases from the town which has made wooden chairs, as a major industry, for as long as anybody knows, and, therefore, it is, by far, the major industry. And, therefore, any disease you picked in that town would be associated with making wooden chairs. I give you that as an example.

Now, in point of fact, I don't want to imply that that chap did not correct for that. So you can use a bias sample of cases and still get a valid answer, which he did; but you must bear in mind that you have got to take care of whether the cases you're looking at are reasonably representative, or rather special, as these were.

Now, there's one form of bias that you've got to be particularly careful of, and that is, diagnostic bias. For example, in cases -- in studies of mesothelioma nowadays, when a pathologist makes a diagnosis of mesothelioma, he doesn't do it with some sort of God-given expertise; he does it partly like that and partly by saying to somebody else, "And what did this man do for a living?"

And if he worked as an asbestos worker, he's more likely to diagnose the case as a mesothelioma than if he didn't. This is the way life works.

So we do have a problem in -- once it is known, or suspected, that there's an association between a factor and a

A. (cont'd.) disease, then there is liable to be a diagnostic bias; okay? It doesn't mean there will be, but there might be. So that's one kind of bias.

And you'll appreciate that that kind of bias will tend to create an association factor which perhaps doesn't exist. I mean, if that hypothesis was incorrect, you would be feeding into your study that preconception; okay.

Bear in mind, in all epidemiology, you can't correct; you just have to bear it in mind.

Controls: we then have the problem of with what do you compare any disease, and -- because, ideally, what you really want to compare it with is somebody who, in every sense, is similar to the person with the disease, had the same opportunity of having the disease but for the factor under review, such as, shall we say, the occupation.

So a good control will be somebody of the same age and sex, perhaps from the same location -- that has to be considered carefully, but in the same location -- who was subjected to the same diagnostic possibilities. So that's one of the reasons you want to pick a hospital control for a hospital case, but not all people in the population have an equal probability of having medical care or of having a diagnosis. So this is the problem.

But you'll appreciate, that is a very subjective and imprecise kind of art rather than a science. The selection of controls is a matter of doing the best you can and applying the best logic you can.

The insecurity comes from the issue of, was it a fair comparison?

Q. Now, still remaining on the case-control methodology, as distinct from its application within a cohort, two potential areas of bias you've mentioned already are selection

Q. (cont'd.) of cases and selection of controls.

It is my understanding that, when taking a group of disease cases and non-diseased controls, and attempting to ascertain their exposure, frequently, case-control methodology employs a questionnaire which asks the person about whether he was or was not exposed, and to what degree. Is that a fair statement?

A. Yeah -- or next-of-kin, yes.

Q. In your judgement, may the use of such questionnaire information present serious problems in ascertaining whether there was exposure and the degree of exposure?

A. Yes.

Q. May I add, ascertainment of exposure as another possible area of bias in the use of case-control methodology.

A. I'd like to make a distinction here, between bias and failure to get a complete story. The -- for example, when I mentioned the diagnostic issue, that is bias; it's not just error. No.

The questionnaire, if administered properly -- and that means, exactly the same questionnaire, under equivalent conditions, to both cases and controls, by an interviewer who does not know whether he or she is interviewing a case or control, will give you information, within limits, that is free of bias; at least, the bias will not be provided by the researcher. There can be bias, still, on behalf of the relative.

I may say, this sounds obvious, but many case-control studies, the interviewer does know whether she is interviewing a case or control.

Q. And that was my final point; that, frequently, there can be observer bias ---

A. There can, indeed.

Q. --- introduced by the use of questionnaires.

A. No question. And in the coding of them, too. I should have gone on to that.

Q. Exactly. My purpose here is not to say that case-control studies are worthless but that they have recognized and controversial limitations which have been discussed a great deal over the last ten years in the epidemiological community. That is a fair statement that I've just made, isn't it?

A. I think it's a fair statement, but I think it's fair to put it in the context that people are beginning to see, equally, that cohort studies have many of the same problems.

Q. Now, the biases which we've been discussing -- and I don't want to suggest that they - well, each of the biases which we've been discussing -- with respect to each of the biases we've been discussing, do you think that they are present, I guess I would say first, generally, when using the case-control methodology within a cohort and then, specifically, with respect to your study?

A. Are case-control studies ---

Q. Yes.

A. --- of mesothelioma ---

Q. I made it terribly -- I made it too complex. Let me try to simplify it.

We've discussed limitations of case-control methodology. When employing a case-control methodology within a cohort -- that is the way you've been doing it; I guess I don't need to explain what I mean by that -- but employing a case-control methodology within a cohort, is it likely that some of the problems of case-control methodology become less likely to occur, I guess, is the way I want to say it?

A. Oh, yeah; you, quite rightly, are drawing attention to the difference between the case-control approach to

A. (cont'd.) analysis of a cohort study from that of a case-control investigation in the general population, such as we have done in mesotheliomas; right? That is a different issue, and all the kinds of problem I've just been discussing will have to be looked at in relation to case-control studies.

But case-control analyses in a cohort are free from all those problems. There is no difference, as I tried to emphasize yesterday -- there should be, if you did a complete case-control analysis of a cohort, you would get precisely the same answer as you get from the man-years analysis, which we've demonstrated, because we've done it both ways. You'll get precisely the same answer.

One of the virtues, can I say, of why this case-control approach to analysis is potentially important -- and, indeed, if we'd known about it fifteen years ago, we'd certainly have saved ourselves a lot of work -- is that, if you carry -- and Mr. Berry, who was here the other day, he may or may not described a study which he's done using this method now.

What it allows you to do is to define your cohort, trace them to find out whether the patients -- the persons died, what they died of; okay. You've got to still do the complete cohort follow-up, and so on. But then what it allows you to do is to turn round, take your cases, select a number of controls, and then go into the work histories.

And that allows you to focus a great deal more work and attention on estimating work history and exposure history than you could if you've got the same amount of resources to do it on the whole cohort.

So, for example, you will find that, in our studies, we have, in fact, up till this 1980 paper, used the brute-force approach of analyzing everybody, finding the work history of everybody, and there's a lot of work in that, and it's taken

A. (cont'd.) a long time.

You will see that now what we're doing in the approach to fibres is to not do that, but take cases and controls, and concentrate on those; so that, instead of having to look at twelve thousand histories, each one with an average of about five different jobs, we now have only got to study a thousand. Now, that allows you, for the same amount of effort, to get more quality, or, again, to do the whole job for less resource. But the answer's the same.

Q. Let me try to separate out a couple of things here, because I think you've answered a lot of questions I was about to ask in the course of that answer, but let me go over the ground just a little bit, so that the record's clear.

In our discussion of case-control studies -- not case-control analysis of a cohort -- we identified selection of cases, selection of controls, problems in the use of questionnaires to ascertain exposure and other information, all as potential problems or pitfalls with case-control methodology.

Now, from the discussion we've had so far, those problems simply don't apply at all, or apply to a very limited degree, if at all, when using case-control analysis within a cohort, such as your own. Is that a fair statement?

A. Subject to knowing what you're doing and being careful.

Q. Right.

A. For example, when you are collecting -- it must be obvious that when you are collecting the case histories of the cases and the controls, the person who does that must also be ignorant of which case and control -- whether they're looking at cases or controls.

Q. Now, in your study, as you've said several times yesterday and today, the two methodological approaches

Q. (cont'd.) produced essentially the same answer, and I take it that, in your judgment, reinforces the meaningfulness of your conclusions. Is that a fair statement?

A. I think this is a general, logical statement, yes; the more times you get the same answer, the more you believe in it.

Q. Now, yesterday -- and I don't remember whether this was Mr. Laskin or Mr. Nelson -- we discussed two possible types of limitation in a study such as your own, and one of those -- and let's take them one at a time -- one of those was the possibility that there could -- their study could be limited in the sense that your comparison group, your cohort comparison group, was the entire population of Quebec. I think we discussed that as a possible limitation.

A. Right.

Q. Does the fact that your case-control analysis within the cohort produced the same conclusion tend to make less likely the existence of a problem with respect to that issue in your cohort; in your study?

A. Yes, I think it does.

Q. Could you explain why.

A. Because the case-control approach to analysis does not -- does not make any reference to the general population of Quebec; it merely looks at the relativities; the relative risks within workers in this industry in relation to their job, to their working history.

Indeed, in our study, and in our discussion of the methodology of this, I think there is general agreement that this is the best method of analysis of a cohort. There's a lot of statistical theory on this, and we certainly didn't invent it, because it doesn't presuppose the comparability of this general population.

A. (cont'd.) However, what it does, unfortunately, depend upon is what has been mentioned several times already: you've got to have some assurance, or some indication, of where, on the scale of risk, does your minimal category lie; and, therefore, the use of both methods obviously strengthened it, because that at least allows you to say that, compared with the general population of Quebec, the low-exposure category -- the lowest-exposure category has such-and-such a relationship. I'm afraid, if you ask me what it has, I've forgotten, but it's about what you'd expect.

This dodges another issue we discussed yesterday: any form of comparison with any other population, you know, why should the people of Asbestos have the same mortality as the people of Thetford Mines, even if they didn't have any industry? You know, we're making all sorts of judgements.

Mortality doesn't really depend, primarily, on occupation; it depends on all sorts of other cultural and economic matters, and so on.

Q. Would it be fair to say that what we've done is, in the cohort study, we've made an assumption of comparability between the population of Quebec and the workers in the mines?

A. Right.

Q. And using the case-control technique to analyze the data within the cohort, we've another assumption of comparability of the workers within the cohort.

A. Yeah.

Q. Would it be fair to say that, as a matter of common sense, that the latter assumption, while not without limitations itself, is fraught with less potential for other variables creeping in to undercut the validity of the assumption?

A. Well, I mean, I feel it's a useful comparison, and I think, you know, with all comparisons you've got to be careful and think about it.

Q. Let me turn to the other issue as well. There was a lot of discussion of the so-called healthy worker effect, and I don't want to cover the same ground on that, but I want to ask whether the use of the case-control analysis within the cohort tends to also undercut the likelihood of that being a problem with your study.

A. Yes; yes, it does. This is one of the things it definitely does do, because they're all workers.

Q. And could you explain, just simply, for the record, why that ---

A. Because -- the problem of this healthy worker phenomenon, which perhaps ought to be -- it is a pity that it's called this, because it really is commonly a healthy worker phenomenon, but not always. What it means is that any working population is different from a non-working population, in some ways; and commonly, particularly in heavy industries, this means they tend to be healthier; at least, at time of employment.

Okay. So that means that, if you compare a working population in a heavy industry with a general population, at least for some years, you would expect them to do better than the general population. And I think they generally do.

But when we -- and you don't know how much allowance to make for that kind of thing. It'll differ in different places.

If you use the case-control approach to the analysis of the cohort, you are looking at people who are all selected in respect of, if you like, being healthy or unhealthy, or whatever it is. They're all subject to much the same selective factors. And then what you're doing is comparing this selected

A. (cont'd.) population in respect to their exposure history.

And, therefore, that comparison -- the relative risks involved in that dodge the issue, to a large degree, anyway, of this selective problem.

Q. So that, by employing the case-control analysis, would it be fair to say that this issue of the so-called healthy worker effect shouldn't be a problem in your study?

A. I think there are more worries than that; yes.

Q. Yes. Now, I've gone through all five of the factors which I started out with twenty minutes or so ago, and I guess you would agree, wouldn't you, Dr. McDonald, that your study is not perfect in that there inevitably may be limitations in the study?

The only reason I keep asking you, is that correct, is so that, if you nod -- do more than nod, so that the reporter will pick it up.

A. Yes; it's imperfect.

Q. At the same time, the limitations which we've discussed over the last day and a half don't, in your judgement, I think it's fair to say, prevent one from using your study and drawing some conclusions.

A. I think it would be criminal not to; yes. I think it's the only data that exists for this industry, and it has used all the available data, really, to come up with the best available estimate; and, fortunately, it seems to have quite a lot of internal consistency. So, yes; I think it is useful information.

Q. Now, I think we all recognize that modesty is a becoming quality, but, at the same time, would it be fair to say that many of your peers have considered this cohort study and all the other features we've discussed to be a model for how

Q. (cont'd.) a large cohort study ought to be conducted?

A. I'd rather not answer that; I don't know.

Q. Now, yesterday, you testified that -- I believe this was in response to Mr. Nelson -- he asked you a number of questions about setting standards in this area, and one of the things that you said was that you would make distinctions between fibres, and that crocidolite, in your judgement, posed a substantially greater risk.

Another thing that you said was, if I understand it, that we ought to attempt to set standards separately for -- on an industry-by-industry segment, provided the data was available. Is that a fair statement?

A. That would imply perhaps a little more than I meant, because I think this is an administrative issue as to what is convenient and sensible and feasible to do.

What I'm really saying is that there is quite a lot of evidence that the -- even if we put on one side the fibre-type issue, which I regard as major -- indeed, I would think that any standard-setting that makes no allowance for fibre type is automatically nonsense. That's rather a strong one, but ...

But if we put the fibre-type issue on one side, then I think it would be again not using all the information available to us to note that there appear to be important differences in level of risk, apparently, in different types of industry.

And we can further say that the direction of those differences is not biologically unreasonable.

Q. While I think you agreed yesterday that your study has implications for and is not unuseful in connection with considering these other industries, that you felt, I believe, that the appropriate data base to be considered in

Q. (cont'd.) judging what to do in those other industry segments was to look at studies of those segments themselves. Is that fair?

A. Yes.

Q. Now, in attempting to look at any of those industry segments, would it be, in your judgement, appropriate to rely on epidemiological studies which attempted to estimate exposure?

In other words, let me try to put the question a little bit differently. We've gone over your study and we've talked about a lot of issues with respect to that study.

For regulatory purposes, would it, in your judgement, be most appropriate, or better, to use epidemiological studies which attempt to estimate the exposures which workers faced as opposed to those which simply attempt to ascertain what level of disease was seen within the cohort?

A. Well, it seems to me irrational that you would have to do that if you want to regulate in terms of exposure. If you want to regulate in terms of exposure, you must use data which includes exposure.

Q. Now, although there are limitations -- and perhaps, as you said yesterday, in 1964, when you had a conference and everybody left that conference charged with the responsibility of going out and investigating not only mining but other industries, I think you testified that you wish they would have all done that.

It is true, is it not, however, that we're not lacking entirely in data for certain of our asbestos-product industries?

A. Yeah; oh, that's true.

Q. We have, I take it, some epidemiological studies which meet the criterion we've been discussing; that is,

Q. (cont'd.) they are cohort studies where exposure was attempted to be estimated?

A. Yeah.

Q. And, again, not asking you in any sense to compare those studies with your own on the five criteria that I started out with this morning, but I take it some of those studies would, in your judgement, be of sufficient quality to shed light on these regulatory questions?

A. Yeah.

I would add here that I would not -- you've asked me, really, to look at criteria of quality, but I believe that, in practice, one has got to use all information available, including bad-quality information.

Q. If we take the product sector of the A/C pipe industry, which we discussed, and putting aside the crocidolite issue, which we've also discussed, would it be fair to say that Dr. Weill's cohort study would be a study which is appropriate for consideration in deciding what to do, from a regulatory standpoint, in the A/C pipe industry?

A. It certainly must be -- it certainly must be one of the studies included, because his is one of the few that did make an effort to measure exposure. It has its problems, mind you, as no doubt he said.

Q. Would the same be true of Dr. Enterline's study, which includes within its cohort A/C pipe plants?

A. Yes; yes. His study has two problems, again, which he would, I'm sure, acknowledge. One is that it is based upon a study of persons who have retired and, secondly -- I could comment further on that if you want me to -- and, secondly, that he was studying a plant in which there was more than one process going on, and it is notoriously difficult to separate the experience of people in different lines.

Q. Dr. Enterline did discuss both of those issues but, at the same time, I don't want in any way to limit you if you have some comments you want to make about them.

A. No, no; the only comment I wanted to make was that -- it's one of the infuriating things, if you like, for professionals in epidemiology -- that people can often get the right answer by the wrong methods. That doesn't mean one shouldn't try always to be, you know, removing the sources of error. And Dr. Enterline is one of the better epidemiologists, at present, in the world, and he is well aware, too, of the problems of looking at retirees.

It might be relevant to say that we have thought it useful to take our cohort and to repeat the analysis (and we've recently presented a paper on this), limiting our data to the retirees. And it is again interesting to find that we get precisely the same dose-response relationship as we get on the whole study.

Now, that doesn't prove that the conclusions from retirees will always give you the same answer as the total cohort. I'm sure nobody would say it was desirable to use retirees if you can use the whole cohort, but at least it provides a little bit of support for thinking that it still is probably valid information.

Q. So, again, I'm not asking you where standards ought to be set; that's not my purpose at all. But it would be fair to say, then, in summary, on the A/C pipe industry, that we have at least two epidemiological studies which ought to be considered when that judgement is made?

A. Oh, yes.

Q. Now, another industry product sector which you mentioned yesterday was the friction materials industry, and I believe last week, when Dr. Berry testified here, he testified

Q. (cont'd.) to a new study by Newhouse and Berry of a friction material plant in England.

A. That's right.

Q. Would, in your judgement, that study be an appropriate one to consider when considering what levels is?

A. It's a very high-quality study.

Q. And it is, likewise, a study which is based upon estimates of exposure in attempts to establish a dose-response relationship?

A. Yes.

Q. Now, we've also discussed another industry sector, and that is the textile sector; we've spent a lot of time talking about Dr. Dement's study.

I'm correct, am I not, that there is another cohort epidemiological study of a textile plant; namely, the study done by Dr. Peto of the Rochdale plant?

A. Yes.

Q. And, in your judgement, would the Peto study be a study which might appropriately be considered in deciding what one might want to do in regulating the textile sector of the industry?

A. They both should; yes.

Q. Both should.

A. Oh, no, no; they both should, yes.

Q. Both Dr. Dement's study and ---

A. Both good studies, yes.

Q. --- Dr. Peto's.

Insofar as there might be differences in the conclusions reached in those two studies, in your judgement, would it be appropriate to try to consider them together to see if there are ways of reconciling the differences; in other words, bring the two more closely into line?

A. I think that, yes -- I think the problem -- I mean, put it this way: if we put on one side, which you have, so far, the whole question of application of insulation, which is another thing, it does look, doesn't it, as if the findings in friction product operations, and in cement operations, if we again put fibre type on one side, and mining are not incompatible. I wouldn't like to say they're identical, but they form a reasonably coherent set of data.

Both Rochdale, on the one hand, and even more definitely, Dement, on the other -- and I did say yesterday that my wife's done a further study of that same plant with essentially the same results -- are very hard to reconcile with those other -- the mining, milling, and A/C pipe and friction.

And, again, we don't have to be surprised, because we're dealing with a number of processes which essentially consist of mixing asbestos; not tearing it apart. And, therefore, it doesn't seem too surprising that the sort of environmental exposures might be similar in their results.

It doesn't, again, seem surprising that, if you go into textiles, where you have the carding, which is one of the most traumatic kinds of process, that the fibre will be different at the end of it.

So, again with one eye on what laboratory scientists find in guinea pigs, and so on, it again doesn't seem surprising that the textile operation might be different.

Whether there is, indeed, a difference in the findings between Rochdale and South Carolina, I still think it needs a lot more study than it's had; it really requires a great deal of careful study.

My guess is that, even statistically, they are compatible with one another; you know, I mean that they're providing estimates which deviate quite a bit, but they both

A. (cont'd.) deviate very much from the other base, so it's not impossible that they are providing this common estimate (one perhaps a bit too high, one perhaps a bit too low, for all I know) of what the textile problem is.

And, again, we can't really directly compare a town in South Carolina with Rochdale in Britain; they are very different environments. So, again, it's not impossible that you might, for other reasons, get some deviation; okay.

So my feeling is, there needs to be very careful examination of those two studies, and that's not just looking at the data already; it would probably mean further work.

And I think what -- if we start looking at what we've got to look at, it is the exposure side of the equation, because I don't think there's any reason to think that, either in Rochdale or in South Carolina, that the data on mortality are seriously wrong. They're almost certainly right.

And if you don't look -- and this illustrates a point -- if you don't look at the exposure, you look at the findings here and you think, "Oh, it might be mines." Instead of looking at the actual figures, you grouped them by low, medium, high, and so on, and looked at the SMR's, you might say, "That's what we found in Quebec."

But what you don't -- what you then find is, you look at what the fibre counts were said to be, and they are many times lower; and that's what got to be looked at, both was the fibre-estimating method appropriate, was the conversion appropriate; probably, I guess it was, or somewhere near it. We could account for some of the difference by opinions on conversion. We wouldn't be able to get rid of it all on that.

So then we come down, I think, to, are the fibres to which persons are exposed in textile operations similar in size and shape; and if that can't explain it, we go back on

A. (cont'd.) another method -- that question was brought up: are there other factors in the environment of a textile industry? I don't know.

Q. What you've said so far, textiles appears to stand out. If we look at mining, if we look at friction materials, if we look at A/C pipes, although you have said -- testified -- that each of them might be looked at separately because they are distinct environmental settings; nonetheless, the data are roughly compatible, in your judgement. That cannot be said about textiles, based on what we now know?

A. No.

Q. And can I sum up by saying that, with respect to any differences between the Peto study and the Dement study, issues which might be looked at in order to attempt to reconcile those two studies would be the exposure estimates themselves, the conversion factors employed in each of the studies, among other things; at least, those two factors are pertinent and relevant?

A. That's right; very close scrutiny of the exposure.

Q. Yesterday, you, in discussing this question of industry-by-industry evaluation, I suppose, you mentioned that there had been a number of -- some asbestos uses which have gone by the wayside. You mentioned children's modelling clay. I think we could add insulation to that group as well; is that a fair statement?

A. So far as I'm aware, nobody sprays asbestos any more.

Q. That was something that happened in very, very substantial quantities during World War II, in shipbuilding, and even thereafter, but now it's no longer a present matter of concern.

Q. (cont'd.) Maybe the legacy of disease from that previous exposure is a matter of concern, but not that industry segment as an existing matter.

A. So far as I'm aware, no; I've not seen spraying for thirteen years.

Q. I take it that, from your remarks, which recommend consideration of each problem on its own merits, you didn't intend, by mentioning the modelling clay example, to suggest that all of the other uses of asbestos which there presently are should fall into the category of modelling clay as opposed to A/C pipe and friction?

You're shaking your head; I just want to ---

A. No, definitely not. I mean, I feel that any part of a regulatory process should, if you like, be intelligent; it should say we're dealing with what is, quite clearly, a hazardous material (some more hazardous than others), it is still maintained to be a useful material; let us at least concentrate on the processes which we really feel are important, and then see what does that leave us with from the standpoint of regulation. That would be my approach.

Q. I have only a few more issues -- at least, maybe a little bit more -- little less organized than some of the ones I've previously discussed, but let me try to take them out.

In discussing your study, we're all aware that it provides, I think, useful information; at the same time, you would be the first to say that it does not provide all information or answer all questions about the relationship between asbestos and disease?

A. Yes.

Q. Would you nod your head. [Laughter.]

Now, a question which came up yesterday with Mr.

Q. (cont'd.) Laskin is the issue of whether short-term high exposure -- burst exposures I think we've kind of come to call them in these proceedings -- whether those exposures might pose a disproportionate risk, as opposed to a continuous exposure having the same cumulative impact.

Now, I believe you testified yesterday that you looked at that question as best you can from the data that you have, and you see no evidence of such a disproportionate effect within your cohort. Is that true?

A. Yes. I say that most diffidently, because I doubt whether our cohort is capable, really, of examining that sort of thing.

If there were a gross effect, we might detect it, but I don't think so; no.

Q. When he was with us last -- week before last, I guess -- Dr. Enterline did testify to having seen, in his cohort, such an effect. He testified that high, intermittent exposure in his maintenance workers did appear to produce a greater amount of disease; and you're aware of that study?

A. I wasn't too aware of that; but I think we can say, at once, that, if there is a difference, that's going to be the direction, isn't it? Again, it is reasonable to think that there are body defence mechanisms, and therefore one can imagine a situation in which those are overwhelmed, as it were, and therefore it's hard to believe that a peak exposure would be less troublesome than the same amount spread over time. It might be much more.

Q. So the investigation in your study, limited in the respects on which you've already testified, should not, in any sense, be considered a refutation of the findings of Dr. Enterline in his cohort?

A. No. No.

Q. Another thing we do not know as a fact -- and I guess we cannot know as a fact, from your cohort -- is what the actual disease is produced at very, very low levels, lower than those investigated in your study. Is that a fair statement?

A. Yes.

Q. That is, what you've testified so far with respect to the issue of what may be happening at low exposures is to say that, so far as it goes, your study indicates a linear relationship between dose and exposure?

A. That's right; yes. It doesn't even do that. I think we have to say that our data, and lots -- other data could be compatible with different models, but it certainly is compatible with a linear model.

Q. When he testified last week, or maybe it was the week before -- it was last week -- Dr. Weill -- I believe it was Dr. Weill -- it was Dr. Weill -- gave us an exhibit which had a number of possible dose-response relationships; one was a straight line to the origin; another was a straight line to a point intersecting the Y intercept; another was a sigmoid relationship; and another was a parabolic relationship.

Would it be fair to say that your data could be reconciled, like Dr. Weill's, with any of those curves?

A. Oh, I think so.

Q. Okay.

One of the things which you discussed yesterday was the biological plausibility of a linear dose-response curve all the way to the origin.

A. Mmm-hmm.

Q. In other words, the so-called one-hit model I think it's referred to.

A. Yeah.

Q. And it's fair to say, isn't it, that that one-hit model and theory originally was developed based on consideration of the effects of ionizing radiation?

A. Yes.

Q. And as I understand that theory, it is that the gamma radiation, I guess it is, penetrates the cell membrane and directly impacts the nucleus of the cell. Is that correct?

A. I don't know.

Q. The assumption, nonetheless, is that the effect is going to be linear as a result of the direct impact on the cell.

Now, one of the things which you were testifying to a minute ago, when we were discussing the issue of intermittent exposure is the possibility of there being clearance mechanisms in the lung, or defence mechanisms in the lung.

Is what you were suggesting that, when there are very large amounts of asbestos inhaled, that the lung is incapable of dealing with that large concentration of dust, and that, rather than all or most all of it being exhaled, a larger proportion of the dust coming into the lung is deposited in the lung?

A. I think that is the kind of thought one has. I think, as soon as you bring up questions of defence mechanisms, you then have to go back and say, what sort of model of behaviour will they follow; are we going to think of a threshold model for those?

In other words, where the body is one hundred per cent able to protect up to a certain amount, and then, thereafter, increasingly fails to do it -- I don't know that this has been studied, but I think you could probably make a case for saying, in general biological terms, that probably not -- probably any defence mechanism is also, perhaps not linear but at

A. (cont'd) least, if you like, increasing -- even if there were a very effective defence mechanism and a low concentration of dust, probably every now and then the odd fibre'll get through.

Q. I didn't mean, in my question, to suggest a defence mechanism is like turning out the light; it's not like you have it or you don't have it. It's a matter of degree. Would that be a fair statement?

A. I think so, yes.

Q. And when we're talking about things which are a matter of degree, like this, it is not implausible, I guess, to make assumptions which suggest that the shape of a relationship may be other than linear. Is that a fair statement?

A. Oh, yes.

Q. Now, we've talked about the clearance mechanism of the lung. I take it that it is, while unproven and maybe unprovable at this point, to hypothesize is unproven and probably unprovable that the lung, at very, very low concentration levels, is able to effectively limit the ability of asbestos fibres to reach the cell; that is not something which we can rule out, either.

A. Oh, no. No, I can't.

Q. So it would be fair to say, we can't rule out the possibility that, in those dose ranges, which your study, by its nature, cannot give us definitive answers, it is not biologically implausible that the relation could be either a threshold or, in any event, non-linear?

A. Yes.

Q. Now, when we were discussing yesterday this relationship between asbestos and smoking, the interrelationship, I believe that you testified -- but I'd like for you to clarify this -- I believe that you testified that your data

Q. (cont'd.) were consistent with Dr. Hammond's conclusion of a multiplicative effect.

A. Yes; they are certainly compatible with that. Yes.

Q. And when you say they are compatible with it -- I know there was a lot of confusion about this, I think, yesterday, because your study certainly implies that you feel that the relationship is additive -- and we may be misreading that implication, so I don't want to ---

A. No; I don't think that. I mean, I would say that the present state of our view on this is that we've no idea; that is to say that there appears to be an interaction. It looks as if it's probably more than additive. I haven't seen any data which is a purely additive model. But it could be less than multiplicative.

And the few sets of data that exist are not able to separate these adequately; nor, indeed, are they able even to begin to look at what is more likely to be the truth, and that is that neither of them apply.

I mean, it would be almost impossible to imagine that two factors will interact in the same way, regardless of things like age of when you start smoking and when you start being exposed.

So, you know, it would seem to me that we're dealing with an approach to the type of model that we're talking about, rather than being able to define exactly what it is.

Q. And maybe -- would it be fair to say that maybe the issue has been posed in too simplistic a way by the coincidence that Dr. Hammond's data were so perfectly multiplicative? Is that a ---

A. Yes; they've managed to produce two perfect, contradictory statements, actually. I mean, before that, they

A. (cont'd.) had a perfect set of data which implied that smoking was an absolute prerequisite, you see, 'cause that's the first model. But that, I think, they would readily agree, was based on the fact they didn't have enough at that time.

So then they have more data, and it comes up with a perfect multiplicative model, though ignoring all questions of dosage of either factor, you'll notice.

And then, now, just when we're beginning to think what a beautiful model that is, they come up with another study which says it looks as if it might be additive after all in another plant.

So, you know, I mean, I'm sure they would be just as humble about the thing as we are. I don't think we know.

Q. Let me just have one final question on it.

When you say that your data are compatible with the multiplicative result, you mean that in a statistical sense; that is, that the statistical significance is such that your data could be multiplicative within the normal statistical confidence lungs?

A. That's right.

Q. Now, yesterday -- I once again can't remember whether it was Mr. Laskin or Mr. Nelson, was discussing the question -- I believe it was Mr. Nelson -- the question of diagnosis of pneumoconiosis, and, as a starting proposition, would it be fair to say that, in a community where there are asbestos workers -- especially a community such as Asbestos or Thetford Mines -- that the a priori assumption would be one that would suggest doctors would be sufficiently acquainted with pneumoconiosis and asbestosis to find it readily in the population where it was there.

Let me state it a little bit differently, because

Q. (cont'd.) I have such a convoluted question here. Would it be fair to say that, in such a situation, where we have asbestos workers, especially as a proportion of the population as large as they are in Asbestos or Thetford Mines, that the a priori assumption would be one of possible over-diagnosis of pneumoconiosis or asbestosis?

A. I don't think I could draw that conclusion. I don't know. I think it would be fair to say that any physician practising in those parts would be liable to think about the possibility; whereas, I expect, if he were practising in the centre of Toronto, he wouldn't think about it; okay.

But that doesn't mean that he will put it on the death certificate. I don't know, you see. I don't know. What you're doing -- I'm afraid there's a lot of evidence that death certificates are very much influenced by your general concepts.

You know, for example, nobody in North America, until very recently, ever put "chronic bronchitis" on a death certificate, 'cause they've been taught at medical school that it didn't exist.

Q. Over-diagnosis is a word which invites comparison -- invites comparison with two possible things that I can think about. One possible basis for comparison is, you know, the truth of the platonic sense -- the sense of, there is asbestosis, and are we finding more than there really is?

Another basis for comparison, a second basis for comparison, would be to compare with what is diagnosed in the general population.

Now, if I take the latter, for a moment, and I ask the question of whether over-diagnosis, as defined as over-diagnosis, as compared to the general population -- would it be a fair statement to say that the acquaintance and familiarity of the medical community in a town such as Asbestos or Thetford

Q. (cont'd) Mines would be likely to produce over-diagnosis in that sense; that sense being the comparison with Toronto or Boston or New York, where ---

A. I don't feel at all convinced it will produce over-diagnosis. I think it is -- there is certainly good evidence for thinking that there might be under-diagnosis in other places; okay.

I think there is a further piece of, sort of light on this; that is that I think one has to consider diagnosis not as an absolute but -- you know, using the diagnoses on the death certificates, how did they, in fact, distribute themselves in relation to the exposure histories?

And that is where I think, if you really are looking for assurance that the error is probably not very great -- that's where you'd find it; because, you know, if asbestosis was being lost in other diagnoses, then you would have to find it somewhere else, you see.

Now, it could be, if you like, that, when we're looking at that small, relatively small, excess of heart disease, that some physicians might conceivably have written those up as pneumoconiosis. Perhaps they would even be right in doing that. But if they did that, then there wouldn't be an excess in heart disease; you see what I mean?

There is just so many deaths, and that's why the overall pattern of deaths, all causes, is something we always need to be looking at, because it is quite possible, through bias, to apparently discover an excess of a disease and yet, overall, no excess; and that could be just due to transfer of diagnoses.

You see, what we do find is a fairly consistent pattern: that the overall excess is, by and large, accounted for by the diseases that it ought to be accounted for. And,

A. (cont'd.) therefore, that makes me think that probably most of the diagnosis is fair.

The kind of errors we do see, incidentally -- and they're, numerically, not important -- we do find, say, some cases of death due to pneumoconiosis, one or two only, in people with little or no exposure. Now, that makes one really wonder. You see, it isn't reasonable.

Now, that is the kind of situation where, conceivably, the physician did it the other way on. Here the man dies, shall we say, from a respiratory disease. "What's your job?" "Oh, I was working in the asbestos plant," so we put down that. So it's this sort of thing.

But it does look as if the diagnostic shift is of a smallish order.

Q. Let me tell you what's troubling me about this, and I'm still troubled, even given your answer.

I'm troubled by what we mean by when we say "over-diagnosis." And I keep coming back to, logically, I see two possible types of over-diagnosis: one is over-diagnosis as compared with the truth, in an ultimate sense, and the other is over-diagnosis as compared with what is the practice in a large population group -- the United States or Canada, or whatever.

Now, over-diagnosis in that sense may be accurate diagnosis in the cohort, but over-diagnosis because there's under-diagnosis in the general population. And I take it you wouldn't rule out that kind of over-diagnosis in your cohort; that is that your diagnoses are good, but that the comparison group reflects an under-diagnosis against some ultimate norm, and, therefore ... Is that a fair -- would you ---

A. Yeah -- I mean, I would put it in a very general sense, yes; that we cannot have any confidence that the diagnostic methods are the same everywhere.

Q. This discussion which we've been having, is that the reason why the standard epidemiological practice is, in cohort studies, to make comparisons based on death certificate information?

A. You mean rather than measurements of illness?

Q. Well, rather than going behind the death certificate and looking at autopsy.

A. Oh, I see. Well, I expect we would rather -- and certainly if everybody had an autopsy, we would at least look at the results, and it might well help.

But one of the real reasons why we can't use autopsy information is because it is biased. We cannot use autopsy information because, within any population, the question of whether you have an autopsy is not a random process; and, secondly, the reasons why you have an autopsy in a certain employed population may be different from why you have an autopsy in the general population. But we know that is so, and that's one of the reasons you cannot -- when we were talking about cohort methodology, it is quite wrong, for example, to correct your diagnoses on autopsy information and use that information to compare with the general population.

DR. UFFEN: Are death certificates ever changed after an autopsy?

THE WITNESS: Yes. I can't be precise about the current state of the law in all our provinces, but, generally speaking, the physician is invited to report the result of an autopsy, when it's available, and then the statistical bureau will correct it in the light of his ---

DR. UFFEN: You would use the revised death certificate?

THE WITNESS: That is so, because the registration authority is correcting it; so that that will be true for

THE WITNESS: (cont'd.) all deaths.

MR. WARREN: Q. So that, can you sum up by saying that, for all of these reasons, in epidemiological studies -- all of these reasons, including, for instance, the fact that your comparison group in a cohort study is the general population -- the appropriate, accepted standard approach is to compare death certificates with general population mortality, since general population mortality is based on death certificates?

A. Right.

Q. Now, that's the methodology which you employed in your studies; correct?

A. Yeah.

Q. And I take it, since that is standard procedure, it doesn't, in your judgement, affect the validity of your study for Dr. Nicholson to have gone out and, somehow or the other, attempted to re-evaluate the diagnoses of asbestosis and say that there was an under-reporting of asbestosis on the death certificates?

A. Oh, no; I think he was perfectly, if you like, at liberty, and a perfectly proper thing to do, if he wished to do it. And what I don't know, because I haven't studied the paper, is whether he then used the corrected diagnosis.

But, I mean, we ourselves did this, too; we looked at the results of autopsies to see what difference it would have made. And, in relation to mesothelioma, we definitely used this, because there was no way of getting at the mesotheliomas through the ordinary death certification process -- at least, there was, but not an adequate way.

MR. WARREN: I think I understand.

Let me ask one final series of questions on mesothelioma. One of the things that you discussed yesterday was

MR. WARREN: (cont'd.) that, as a matter of general population statistics in North America, that, during the 1970's, mesothelioma rates had been rising in males at a rate of, I think you said, five to ten per cent.

Q. Is that correct?

THE WITNESS: A. This is my best available estimate of the kind of rate of increase that appears to be going on, in males.

Q. Now, given the latency period for a mesothelioma, would it be fair to say that these increases in the rate of mesothelioma in males is, in your judgement, a legacy of past high exposure?

A. A legacy of past exposure; how high? I mean, you know, if you want me -- I think it's a legacy of past amphibole exposure; okay?

Q. Okay. But that it reflects what happened four years ago, as I guess ---

A. That sort of time.

Q. Another implication -- let me ask if we can draw this implication -- the rate of increase which you quoted is a rate of increase in males; not in females?

A. That's right; that's correct.

Q. Would it be fair to say that, because it is males, predominantly, who have been asbestos workers, that the increase in mesotheliomas is reflected occupational rather than non-occupational exposure?

A. Yes; definitely.

Q. In fact, would it be fair to say that the fact that rates for women have remained roughly constant would suggest that non-occupational asbestos exposure, especially apart from that exposure in the home of an asbestos worker, is not a problem of increasing concern, in any event?

A. That is my interpretation of the information. It does tend to suggest that the contribution of occupational exposure is the main factor affecting the increased rate in males, and the absence in females implies that the non-occupational exposure is probably not having any changing effect.

There may have been some low level before, but the constancy of this suggests that non-occupational exposure is not having any important effect -- in North America.

MR. WARREN: Thank you, Dr. McDonald; and, Mr. Chairman, I'm finished.

DR. DUPRE: Thank you.

Who is next in batting order?

MR. CASGRAIN: I think I am.

DR. DUPRE: Mr. Casgrain; proceed, please.

THE FOREGOING WAS PREPARED
FROM THE TAPED RECORDINGS
OF THE INQUIRY PROCEEDINGS

DEREK WEST

CROSS-EXAMINATION BY MR. CASGRAIN

MR. CASGRAIN: Q. Dr. McDonald, you stated in the course of your testimony, and correct me if I'm wrong, something like the further away from the mill the fiber goes, that is when it gets into the manufacturing system, the more susceptible to penetrating the lung and remaining there it becomes. I think that you stated that this was because of the cleavage of the fiber, and that it then became more susceptible because of its confirmation to penetrate the lung and remain there...at least the defence mechanism of the lungs were not as capable of dealing with it as with the fiber. Is that correct?

THE WITNESS: A. Yes. I think in reflection... I certainly agree that I said something to that effect, and on reflection it was, I think, a slightly oversimplified statement, because I think you could say that moving into brake linings is quite a long way from the mine and mill, too, and perhaps what I should have said is the more processing that goes on that it is likely that the fiber will be broken down by cleavage and that then I would make another point...not that it would penetrate better, but that it may not penetrate better, it may simply be a matter of a more serious biological effect of the finely-divided fibers.

I'm going beyond knowledge. I am rationalizing what we observed.

Q. Except that you also stated in the course of your evidence that the longer the fiber, the more biological effect it has.

A. To the extent that it can get there, yes. But the trouble is, we have...you see there, if you have a very long fiber, it can't get there, so it can't do the trick.

I think there is experimental evidence here that even at the cell level there is an increase in the

A. (cont'd.) biological effect up to a certain level, but I think it's got to be, you know, beyond a certain level probably this isn't true.

Q. Of course, we are talking about fibers that remain longer than five microns, aren't we?

A. Yes.

Q. And that have a diameter which is smaller than one micron?

A. Yes.

Q. Otherwise, as you know, it wouldn't be the same thing.

I think evidence has been made here...I wasn't present, but I think it was demonstrated here that there is a mechanism called macrophages that actually may or may not be able to fight and the length of the fiber has something to do with this?

A. Yes.

Q. May I refer you to tab number eight of exhibit nineteen, which is the paper by Dr. Nicholson.

A. I haven't got that.

Q. Have you not got that?

A. I wonder. I don't know that I have.

Q. I think it was filed yesterday as...

A. Oh, I may have it. Just a minute.

Q. It is identified as tab number eight of exhibit nineteen, which is not the one you have before you.

A. Yes. Which page?

Q. Have you got that?

A. Which page?

Q. Page nineteen.

A. Nineteen, yes.

Q. I would like to quote, or you to read, the last part of the last paragraph on page nineteen...line five, it is.

Q. (cont'd.) Starting..

A. Starting "an alternative source"?

Q. Yes. "An alternative...", that is the paragraph I'm talking about, and I'm going to line five, which starts, "During application or removal of insulation products, it...", meaning the fiber..."is further manipulated and the fiber is reduced in length and diameter. As these smaller fibers can readily be carried to the periphery of the lung, penetrate the visceral pleural and lodge..." etc., etc.,...

A. Yes.

Q. "...they may be of greater importance", etc., etc.,

A. Yes.

Q. I am wondering here when one talks about reducing the length whether you agree with that statement in the light of the fact that it is established, I think, that we are...five microns is of some importance. Do you agree with that statement?

A. I think that...well, what I think happens, and I'm not an expert, what I think happens is that the fiber is broken down and that by its nature it is more likely to be divided in width than it is in length.

Now, that doesn't mean it won't be broken in length, too, but it will be broken more in terms of cleavage.

Q. But if it is reduced to a length which is smaller than five microns, then what is the result? Is it not that macrophages will be better able to deal with it?

A. No. If it is smaller, in many respects it will penetrate better. But it does seem that there is some relationship between the total quantity of fiber, in the final analysis, in relation to the biological effect. There is quite a lot of controversy still about what is the importance of the very tiny particles and fibers.

Q. My question, Doctor, I'm not...

A. I'm sorry. Have I missed your question?

Q. Well, perhaps I misunderstand you. I thought, I took as a base the fact that when we were talking about...shall we say harmful fibers...they had to be longer than five microns?

A. Oh, no.

Q. No?

A. No, no. I think the harm is in relation to the length. The longer they are, the more harmful.

Q. That is right..

A. Within limits.

Q. Longer than?

A. Yes.

Q. But if they are shorter than?

A. There is not a cutoff point of five microns.

Q. No?

A. No, no. I mean, it is convenient to measure the ones more than five microns, but I don't think there is any evidence that damage stops at five microns. No, no. It wouldn't even be reasonable to think it. I think it probably is a matter of increasing with increasing length.

Q. Fine. If they reduce in length, there is a decrease, is there not?

A. If it's reduced in length, so long as it's a reduction in length that matters. In other words, I am presupposing...and I must say I am beyond my area of knowledge... I am presupposing that the biological range of effectiveness is important up to a certain amount. Beyond that it perhaps is no...there is no more damage done by, shall we say, something that is thirty microns or a hundred microns. It could even be that there is less damage because the cells can't even begin to deal with the very big ones. I don't know.

But within the range which these scavenging cells

A. (contd.) can deal with it, it looks as if the longer they are, the more the probability of trouble.

Q. Yes, but five microns was not just picked out of the sky. I mean...

A. Pretty well.

Q. Yes?

DR. UFFEN: Is my recollection correct that the five microns originally came about because it was the limitation of the physical measurement?

THE WITNESS: I think this is correct. I can't say...

DR. UFFEN: Below that, they couldn't count them?

THE WITNESS: I think that's what it amounts to.

MR. CASGRAIN: Q. I suppose I didn't read my material properly, but I thought that perhaps the length had something to do with the ability of macrophages to actually move around the fiber, and eventually it could be...it would go out in the sputum, is that not correct?

THE WITNESS: A. I think you and I have similar levels of knowledge on the subject, I really do.

Q. I will take this as a compliment.

A. I don't know. I really don't know. We are now outside...I really would not want to discuss what is a matter of experimental toxicology. I don't know. This is my impression of what they say, yes.

Q. It was my impression, too, from what I had heard and read that in effect your macrophages were better able to copy with the fiber because as it remained there in the lung, they were able...so they said...

A. Yes.

Q. ...I suppose as a result of experiments, to actually go around the cell and then it would be expelled by the...

A. That's right.

Q. ...but then I suppose that, as you say, we ought to go back to our own articles and read them.

A. That's right. I think we have...

Q. I would like to put myself in the same class as you by saying you and I should go back and read our stuff, but perhaps this is..

A. I think we have a kind of Walter Disney concept of, you know, the cell grappling with this fiber which is too big for it. I don't think we really know.

Q. While we are on the subject of the cell, may I talk to you about some other matter dealing with the cell? Most of the data that we have on the biological effects of chrysotile asbestos, I think, can be divided in two categories. There are epidemiological surveys and biological experiments in animal models and tissue cell resistance. What I would like to address myself to at this stage, if you wish, is to the issue of the chemically altered asbestos by the industrial process. I am no longer dealing with the cleavage. I am dealing here with the fiber which has been incorporated into, for instance cement or resin. Do you have any information in this respect, Dr. McDonald? Or any knowledge in this respect?

A. No, not really. I mean, the only thing I know is that it does happen. That is, the quality of the fiber is changed by things like heat, by exposure to solvents, to acids and so on. So for example, I think there is evidence that fibers that are incorporated in brake linings are exposed to intense heat and there is a degree of what they call denaturing of the fiber. I mean, the fiber's quality tends to disappear under heat.

So, yes, we do know that and we do know that asbestos fibers are mostly very absorbent substances...that's one of the reasons they are used...and that therefore in an asbestos cement mix, it is likely that particles of cement will adhere all along the fiber. I mean, all these things, I think, have been

A. (cont'd.) observed and there have been hypotheses that this affects the biological result. I don't know.

Q. But you do not know yourself of any studies that you...

A. No. Not really.

Q. There have been studies, but you would not know of those studies?

A. Not that I would wish to express an opinion on, no.

Q. You stated in the course of your evidence, and correct me if I'm wrong...yesterday, I think it was...you talked about safety which was readily achievable if not achieved already. Do you remember saying that?

A. That's about ten years ago?

Q. No.

A. More recently than that?

Q. It would have been yesterday.

A. Oh, I see. But I can remember writing it about ten years ago.

Q. All right. Would you emphasize on this for me?

A. This is in the mining and milling industry?

Q. Yes.

A. Yes. That the evidence that we have on the chrysotile mining and milling industry suggests that at most current levels prevailing we are talking...I don't know exactly, but I suppose we are talking of levels which for the most part are around about two fibers or less...maybe one fiber or less...in many parts of the industry. There are a few exceptions. There are a few places in crushers and things where it's difficult to achieve this and where fortunately for the most part workers don't have to be exposed, and if they are exposed, they could take special protective measures.

A. (cont'd.) Okay, so what I'm saying is that such levels...these sort of levels are now being achieved in the industry and so far as we know about exposure response in this industry, it certainly means that we would certainly never be aware of any hazard. That doesn't mean there isn't one. That's what we've said all along. We have to extrapolate as to whether there is any hazard at that level, what the dimensions of that hazard are, from what the prevailing estimates of concentration are.

I don't know if that answers your question.

Q. Yes, that's fine. Yes.

I think you also spoke yesterday, although briefly, as a passing reference to the case of an asbestos worker in the mine or the mill, of age...I think you mentioned sixty or sixty-five, who would have a certain fibrosis. You didn't mention any percentage of incapacity as a result of it, but you talked about a fibrosis, and that could he or maybe he should remain in the mill and work, provided of course the levels of exposure are those to which you just referred.

Am I correct?

A. I'm not just quite sure. I think perhaps this was in the context of should a physician advise a worker who has some evidence of...

Q. Mmm-hmm.

A. ...exposure, to leave the job. My feeling is that he should discuss the findings with the worker and explain it to him. I certainly would feel that an automatic saying - you have trouble, you have trouble in your lungs, you should leave the job or you should stay in the job - would not be the right answer. I think that the doctor should say, I see this, this is what I believe is the implication for you.

My guess is, that particularly at present levels of exposure in the mining and milling industry, that a worker who has got some evidence of exposure, and particularly

A. (cont'd.) if it were not incapacitating him, would probably almost certainly choose to stay at work, because he will be paid a full wage, whereas if he leaves his job he will have great difficulty getting a job.

But, you know, I see this as a decision which is... it is not dictated by medical factors. It is dictated by what is in the worker's opinion of what is in his own interests.

Q. Perhaps in the same vein, you spoke as well, and I think this morning my friend came back a little in the same matter, in respect of detection, of early detection of asbestos-related diseases. I am talking, for instance, about the mines and mills, and you made a distinction between sensitivity and specificity.

A. Right.

Q. I'm afraid I didn't quite follow you when you mentioned it. Would you please explain to me again?

A. Yes. It is, briefly, that if you have a test which is very sensitive, is capable of picking up very tiny changes in some appearance on the x-ray or function tests, then almost for certain that test will not be very specific. It won't tell you what the change, the depression in norm, the depression is due to.

Q. Oh, I see.

A. It might be due simply to the weather or the fact that he had had, shall we say, a respiratory virus infection that passed through the community, or something like that. It would be very nonspecific. All you could say is that this group of workers compared with their observations, let us say, six months ago, have got a detectable depreciation, deterioration in function. It would be very sensitive, but it wouldn't tell you what. It would alert you, right? It would alert you.

And this is the problem, that really early detection methods don't say here is a man with asbestosis. What

A. (cont'd.) they say is, here is a man who conceivably might have asbestosis, the early signs of the effects.

Q. So that you recommendation would be that you should not only look at the whole group and look for sensitivity, but you should also have as many...repeat the examination in a regular fashion?

A. Within reason. Within reason. I mean, I think we have to be a bit cautious. I mean, I think the concept that used to be acceptable of x-raying peoples' chest really very often maybe ought to be reconsidered. We have talked a lot about the interaction of smoking and asbestosis, but we haven't talked at all about the possibilities of interaction between irradiation and asbestosis. You see, it isn't impossible at all that we actually would be increasing infinitesimally the chance of getting lung cancer, as a result of x-raying people.

So you've got to balance benefit against potential cost here.... as always. I mean, we were talking about risks. Everything has its risks. Even having a chest x-ray.

Q. Even the x-ray machine has a risk for the worker?

A. X-raying a person to protect him will carry a risk, so there it is.

What I think needs to be said here is, that it used to be, particularly in mining industries, a sort of prevailing view that one of the ways you protect the worker is by periodically examining him and x-raying him for early signs of trouble, and then taking him out.

Up to a point, this still makes some sense. But it clearly, in relation to asbestos, is not a satisfactory approach because that way what you would be doing is picking up people, shall we say, after ten years exposure, who maybe would never

A. (cont'd.) develop any important degree of asbestosis if you detect it early, but you would be allowing them to accumulate ten years of exposure which would carry ten years worth of risk of cancer, and you would not prevent that by withdrawal. So I think it does mean that in terms of supervision of the worker, we must not rely on the concept of early detection and withdrawal. It should be seen more as a method of monitoring for evidence of failure to control the environment.

Q. Talking about x-rays very briefly, I think we discussed bias and possibilities of overdiagnosing and so on. When one reads x-rays, there is a method to reading x-ray, is there not?

A. There is.

Q. Is it true to say that it is almost a specialty when you talk about reading x-rays of people who have been affected or may be affected with asbestosis?

A. Yeah, it is too specialties. We've got on the one hand the normal diagnostic skills, the ability of the radiological specialist to be able to look at an x-ray, take into account the results of the physical examination and all that, and say I think this man has or has not got a degree of asbestosis. That is a clinical judgement...the normal medical judgement of skill.

In epidemiological surveys, and this applies to really monitoring workers, that type of diagnostic...

Q. Sorry, if I just could make sure. You said this applies to monitoring? You are not talking just...

A. I'm sorry. But when we talk about...I've now given you an example of what you might call the orthodox clinical use of x-rays, where we use specalist radiologists who are not only specialists in observing, but in interpreting.

Q. Right.

A. When we come to survey work, and that includes

A. (cont'd.) monitoring, we are not then concerned with interpretation. Indeed one of the worst types of reader to have when you are doing survey work, is a radiologist. He cannot get away from his training, which is interpretation.

The ideal reader would be an x-ray scanning machine that didn't think at all. What you really want is something that will measure mathematically the opacities in the lung. That's what you really want and there is work going on now which is beginning to be able to do this.

Okay, so you see in the development of the technology of the use of the x-ray for preventive purposes and for epidemiological purposes, depends upon highly-standardized technique and highly-standardized recording of what is seen. Not thinking or interpreting, recording.

And indeed there is a lot of evidence that you can, short of this scanning machine, that you will do best by picking not experts, but observant people who can do that, who have the skill to observe and to record what they see without thought.

Q. To avoid bias?

A. To avoid the bias of interpretation, yes.

Q. You would do that, perhaps, by introducing a number of x-rays, some of which would not come from asbestos workers, and some would?

A. You must. You must do that.

Q. You must do that as you do the reading?

A. You must do, it must be done blind, it must be done independently. You must not, for example, have what is sometimes called consensus reading...very useful in clinical circumstances for the three or four radiologists to gather round an x-ray and discuss the meaning. That is a very useful process when you are trying to decide what is wrong with Mr. Smith, but it is not a good process when you are

A. (cont'd.) trying to assess in a reproducable way the level of prevalent changes in the working population.

So it's a different kind of expertise that we are talking about.

Q. Listening to you, I'm beginning to wonder whether your pneumoconiosis committees that you would have in the Workmen's Comp shouldn't be changed from time to time?

A. I think the Workmen's Compensation problem is a diagnostic one. As I see it in a Workmen's Compensation situation, the panel, the experts, are being asked as physicians or experts, what do you think of this patient, what do you think is wrong with him, first of all, and then in relation to his work to what extent do you think his work caused it. And that's not what we are considering when we are monitoring a group of workers, or when we are doing epidemiological surveys.

Q. Would you say the same thing of rales?

A. Of?

Q. Rales?

A. Rawls?

Q. Rawls? Some pronoune it rawls, some pronounce it rales.

A. Yes, yes. I'm with you.

Q. Would the English be rawls?

A. Rawls, yes, rawls.

Yeah, I would I think, now I come to think of it, because we've just been doing a survey, for example, in dust-exposed workers, in which we are trying to get away from the person listening through the stethoscope. We are recording the noises with a tape recorder and we are analyzing them with a computer.

Q. That's in the form of an experiment at this time, is it?

A. It is, yes. But there is some basis for it.

Q. That could give far more certainty than just a person...

A. Well, a lot more objectivity.

Q. Yes.

Dr. McDonald, using the ILO classification, if you read an x-ray and you find abnormalities.....by the way, before we go any further, I may be wrong. Perhaps the translation may be my problem. I hear the word anomalies and abnormalities. Am I making a mistake, or is that...am I translating?

A. I think...anomaly...I think actually the word in English is changes. I think, in fact, the ILO classification, it's important to emphasize, is not a diagnostic classification. To some extent, unfortunately, it is sometimes used as a diagnostic classification.

You see, what the classification does, is to provide a framework for describing the opacities in the lung as seen.

Q. Yes, as seen. Could we stop there for one second?

A. Yes.

Q. Doing this, if you find opacities to the extent of, say thirty percent, how would you, as a reader...if you were reading, qualify that?

A. One doesn't use the term thirty percent. One is classifying the changes in terms of...on a scale...

Q. Zero?

A. ...it's a twelve-point scale, from zero zero dash up to three, four. It is a continuous scale that you are classifying on, and you are classifying against, by comparison with standard films. In other words, you have got these standard films which provide you with the levels and you are comparing each film in respect of the standard, and deciding what the score is.

Q. All right, fine. I think I made a mistake

Q. (cont'd.) in my question.

If you saw zero slash one, for instance, using the ILO classification, would you classify that as a change?

A. Yes, we would. Yes, it is a change because it is a change from zero zero.

Q. Mmm-hmm.

A. But the nature of the classification is that the group zero means essentially you believe there is no change.

Q. May I refer you now to tab eight, exhibit nineteen...sorry, eighteen, I think it is...tab eight, page 361.

MR. LASKIN: Your own...

THE WITNESS: A. Page...?

MR. CASGRAIN: Q. Eight, sorry.

A. Eight, yes.

Q. Page 361 of tab eight.

A. Page 361...is this the lung function one?

Q. Yes, yes. What it refers...really what I'm getting at is the following: I think in that table at page 361, you talk about domestic cases. That would be...

A. I'm sorry, I'm still not with you. Epidemiological surveillance paper?

Q. Yes. Page 361.

A. Page 361, yes. All right.

Q. The table five, which is there....

A. Yes.

Q. In it we see, and it is referred to in the article and you mentioned it in your evidence as well, that you talked about cases where they were domestic cases. That is, either daughters or sons of workers...

A. Yes.

Q. Correct?

A. Yes.

Q. Now, obviously you do not have specific exposure data on these people, only to say that they were in

Q. (cont'd.) contact with...

A. That's right. Correct.

Q. May I, in this respect, ask you Dr. McDonald, at what time, for instance, these would have occurred? What period...?

A. I see. I'm sorry, I would need to look in detail at this paper, but in general we excluded occupations...if I recall right...within ten years of death. If we didn't we should, and indeed we normally do that. The point being that we would not include exposures which, from our general concepts and beliefs, would not have contributed to the disease.

Q. All right. But assuming you did, the exposure we are talking about would have occurred in what year? Would it be nineteen...

A. We would have that recorded. It could be anything before then. It could have been anytime...

Q. Before what year?

A. Anytime before this cutoff point, which as I say I'm guessing was probably ten years. I could look it up.

Q. I'm really looking for 1930 or 1940 or 1955?

A. Oh, I see. Well, it's not recorded here because each one will be different.

Q. Do you have any idea?

A. No, because this is all cases, if I remember, this is all cases ...I think this paper is based upon all cases, 1968 to 1970, dying 1968 to 1970, and I am now guessing if they did 1968 to 1970 and their average age was around sixty, let us say, then probably the relevant exposure was somewhere around about 1935 to 1945, something like that.

But, I mean I can't say. I would need to look all this up, but that's the kind of thing.

Q. At that time, I think, in the mines and mills in that area...

A. These were not in the mining and milling area. These cases are across Canada.

Q. Sorry, maybe I didn't read properly. I'm reading here table five.

A. Ah, well now if we go back to table five, that may be...

Q. Mining and milling.

A. Okay so we've then got three cases, mining and milling.

Q. That's right.

A. That's right, yes.

Q. Those are the ones I'm concerned with.

A. Within the seventy-odd cases, yes.

Q. All right. At that time...that is, around the years you just mentioned, the conditions in the mines were such that the workers came home with their clothes actually full of fibers?

A. Oh, yes.

Q. That would happen every day?

A. Oh, yes.

Q. In fact, weren't some of the homes even built on asbestos tailings?

A. Yes.

Q. So that there would be the dust as well from the foundation, the very foundation on which the house was located?

A. Oh, yes. The general environmental exposure was quite high even much more recently than that.

Q. So that with respect to those people mentioned in table five here, the exposure that they would have had as a result of the contact with the workers, could you now guess at

Q. (cont'd.) the number of fibers?

A. No.

Q. It would have been higher though?

A. What I can't guess at all, I could guess...no, I couldn't guess at anything. But what I could say is that concentrations of fiber in the homes of asbestos workers can be very high, because all the soft materials accumulate fibers and then as you shake them or whatever, it just puts out a dust which could be quite high. I mean it could be ten fibers, it could be really quite high.

Q. Of course we are talking about at that time, where it would have been very high?

A. Yes, it would be quite high. It could be...you know, as long as this kind of process went on, it could be high.

Q. Turning to another subject...

MR. CASGRAIN: Mr. Chairman, I have only one question left. I am just wondering whether...if you have any plans for this afternoon. Because I can either adjourn and we can go back this afternoon, or...

DR. DUPRE: Well, we will be going...for the information of the other parties...we will be...our magic hour is four o'clock, which is when Dr. McDonald has to leave.

Now, bearing that in mind, if you feel we need say two hours this afternoon, we should then certainly be back here by two.

MR. WARREN: Mr. Chairman, I will be very brief, I would think, in the order of about ten or fifteen minutes.

MR. GIBSON: My questions probably run about twenty-five minutes.

DR. DUPRE: Okay, and who else will be coming to bat this afternoon?

MR. McNAMEE: Five to ten minutes.

DR. DUPRE: Okay. Mr. Ublanski?

MR. UBLANSKI: I may have five minutes.

DR. DUPRE: Okay. I think it looks containable to the two hours, so perhaps on that, Mr. Casgrain, if you want to put your last line of questioning, or did you anticipate that would take more than about ten minutes?

MR. CASGRAIN: No, because this may be one question, again, where Dr. McDonald and I are in the same...

DR. DUPRE: Okay. Why don't you proceed.

MR. CASGRAIN: Q. I would like to turn to the counting of fibers, and I say that because I think perhaps you and I may decide not to discuss it, but you did say yesterday you were not an expert in the counting of fibers?

THE WITNESS: A. That's right.

Q. Nevertheless, I would like to ask you the following question: When you are counting fibers and you are down to the level of two fibers, what is the margin of error, in your view, do you know?

A. No.

Q. No idea?

A. No, I really don't. There won't be a single answer to that question. It will depend upon the counter and the conditions. You know, the sort of...I say conditions...if you are counting in a situation where there is a stable level, you'll have a greater degree of confidence than if you are in a situation where they are fluctuating. Again, the skill of the operator. I'm sorry, I couldn't tell you.

Q. You can't help me on that?

A. I couldn't give you a measure, no.

Dr. Gibbs later might well.

MR. CASGRAIN: I have no further questions, Mr. Chairman, at this time.

DR. DUPRE: Shall we adjourn for lunch and return at two o'clock, precisely?

Thank you, Dr. McDonald.

THE WITNESS: Thank you.

THE INQUIRY RECESSED

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THE INQUIRY RESUMED

MR. LASKIN: I think we are ready to proceed, Mr. Chairman.

DR. DUPRE: Thank you, counsel.

Who is next in the batting order? Mr. Gibson, I believe? Would you please, sir?

DR. GIBSON: I am. I am up and I'm out right immediately because I don't have any questions, thank you very much, Dr. Dupre.

DR. DUPRE: Oh, you'll go after that, Mr. McNamee? Thank you.

Sorry, Brian Gibson

MR. GIBSON: Yes.

CROSS-EXAMINATION BY MR. GIBSON

Q. I would like to explore just a few points with you, Dr. McDonald.

The first thing I would like to see if we could possibly tie a few things together. There seems to be a small excess of heart disease in your study, and generally the SMR's running, oh, around one or maybe a little bit above that.

My understanding is that usually when you take a look at a worker population, you see heart disease as one of the areas where you see the SMR lower than one... if you are

Q. (cont'd.) dealing with a population where it requires fairly healthy workers in terms of the work that you are doing, and also that if there is some experience in the population that is increasing their risk, and the overall SMR isn't much greater than one, then some of your other major causes of death should show up with an SMR lower than one.

Is my understanding of that correct?

A. I understand your question. I'm not sure that I would go along entirely with your statement that other studies tend not to show this sort of thing. I think it may be true of situations where one is looking for relatively earlier effects. I mean, if we what we are seeing here is this effect in persons with twenty or more years, that group in fact is, shall we say thirty years on average, of work, and in the presence of a disease process which is known to be very long and insidious, I don't think that necessarily applies to all occupational studies.

You see, I mean here we are looking at disease... several diseases, one of which is a chronic fibrotic disease, and the other are the cancers.

I don't know that there are many occupational studies where you are looking at both those kinds of hazards.

I mean, you could be right. I'm just raising this issue. I'm not surprised, personally, about what we observed.

Q. In terms of the...what I'm wondering is, referring back to the question that the Chairman raised yesterday... is whether there might be some effect of misclassification taking place here, that this could be one explanation for the SMR for heart disease being a little bit higher than you would expect it to be?

A. I think it could be a composite of that. I mean, I recognize the real possibility of a degree of

A. (cont'd.) misclassification there on the one hand, and the other general point that I was making earlier that people die from a variety of conditions which collectively reduce their chance of survival, and to a degree it's a matter of judgement or luck what gets put on the certificate.

In other words, if you have heart disease and you also have some degree of impaired pulmonary function, your changes of dying of that...shall we say coronary...are less than if you didn't have it.

Should that be ascribed to asbestosis, should it be ascribed to the coronary, I don't know and presumably the certifying physician often doesn't know either.

I think it's a contamination both ways, yes.

Q. I'm trying to explore just a little bit further the possible directions of the bias that might be operating here. That given a situation in which a vast...heart disease is a very common condition...I gather about a third of the population die of it...that also in a population in which asbestosis is considerably more common than...is a fact of life...that...and certainly would have been in terms of what past exposures were like, that it would not have been a big deal to...in terms of a fellow who died in his home, or whatever, to say well, he died of a heart attack or he died of asbestosis. It would not have been of great concern to many of the certifying physicians, before it became a matter of epidemiological importance, to get that information correct, what they put on that certificate?

A. That's right. That's right. I agree with you completely. As long as you don't mean in a way that there is a correct answer?

Q. No.

A. I mean, you say 'not get it correct'. I'm not sure how you would ever get it correct.

Q. I was trying to suggest that simply because this is a common disease that people could diagnose, and that the physicians would be very familiar with, wouldn't necessarily mean that this is what they would put on the certificate.

A. Yes.

Q. And that the thing that might suggest that there is a direction in the bias, if anything, is that the SMR's for heart disease are slightly...you know, perhaps higher than you would expect...in a working population?

Is that a fair statement?

A. I would be interested to know...and I'm asking you now...whether the same sort of thing is observed in coal miners.

Q. I don't know.

A. I think it is, you see. And I think that here again, you see, I think that if you have...after all, the cardiorespiratory is sort of one system, and if you've got a stress on one part of it, I would expect...I'm sorry, I don't find it surprising. I would expect that we would...it ought to have some...reflect itself to some degree in an increase in heart disease.

Q. But in some sense if this would be due...should be related in some ways to the asbestos exposure?

A. Oh, I think it's all part of the sum total impact of the exposure, yes. Oh, yes.

Q. If I could turn for a moment to tab twenty-two, page 814, this is...tab twenty-one, sorry, page 814.

It's the equation for the relative risk.

A. Yes. Which page are you looking at?

Q. Page 814.

A. 814? Yes, right.

Q. The relative risk equals one plus point zero zero three eight...

A. Yes.

Q. ...times the number of fiber/ML years.

A. Yes.

Q. This is basically drawn from the case control analysis of your cohort, I believe?

A. It's drawn from this particular one, yes. Yes.

Q. They were matched for smoking in this particular analysis, the cases and controls?

A. I think they were. I can't remember whether this particular formula used...I think this was on the smoking, matched.

Q. My understanding of the implication of the formula that we are talking about a one point five percent increase in the risk to the worker over his lifetime for lung cancer, per fiber per mil in his exposure. So that if that's added onto his risk from smoking, if he was a smoker, and if he's a nonsmoker, then it's added onto his minimal risk as a nonsmoker?

A. Except it's not exactly added. It's, if you like, multiplied. It's an increase...it's important to bear in mind that this percentage increase is calculated against what his risk would have been if he hadn't been exposed.

Q. Yes.

A. So fortunately, we can say because of the nature of the tobacco / asbestos interaction, that broadly speaking that same kind of equation will apply whether a man is a smoker or a nonsmoker...for practical purposes.

Q. Mmm-hmm.

MR. LASKIN: I'm sorry, I didn't mean to interrupt, but could I just clarify something here, because I'm not sure I've understood it.

I originally thought that the original line for relative risk which you got, which appears on page 812, was

MR. LASKIN: (cont'd.) the line that Mr. Berry fitted from your total cohort analysis...or your total anlysis in 1975?

THE WITNESS: A. Yeah.

MR. LASKIN: The conversion ratio is the ratio you got from your two hundred and forty-four subjects in matched controls, one of which was for smoking, and then you applied that conversion ratios in fibers, back to the original line that Mr. Berry had fitted.

I just wanted to be...

THE WITNESS: I think both are true. I'm sorry, I would need to study it to know exactly which this one is. It would not make any difference.

MR. LASKIN: One way or the other?

THE WITNESS: Yes.

MR. LASKIN: I'm sorry, Mr. Gibson.

MR. GIBSON: Q. Okay. Which means that we are talking about someone exposed to one fiber per mil, over a lifetime, working lifetime?

THE WITNESS: A. That's right.

Q. You are talking about a relative risk...

A. Forty year, yes.

Q. Is really one point zero one five?

A. That's right.

Q. I believe you mentioned yesterday that for textile workers in South Carolina, their risks were turning up at one to two orders of magnitude greater?

A. It looks like it.

Q. So that you are talking about ten to a hundred times greater risk?

A. Possibly. I mean, I'm still...would prefer not to put too much on that one, because it's a very new set of data. The Rochdale information has been much more lengthily

A. (cont'd.) presented and discussed, and would also point to a fairly substantial increase....maybe an order of magnitude greater.

Q. Which would...if I'm playing with the figures correctly, an order of magnitude or ten times would make a relative risk of one point one five?

A. That's right. That's what it would imply.

Q. In that industrial situation?

A. Mmm-hmm.

Q. And if in a specific industrial situation, if in fact it were two orders of magnitude greater, it would mean a relative risk of two point five?

A. It would, but I don't think even the South Carolina manages two orders.

Q. Okay. If I could take a look at, with you at tab seventeen, page eight, figure four, and this is the graph where the equation of the relative risk for smoking is plotted against the relative risk for asbestos exposure?

A. Yes.

Q. That...and I believe when we were talking about it yesterday you were saying that one fiber per ML over a lifetime would be half a cigarette a day, or perhaps a cigarette a day?

A. Something like that, yes.

Q. So that we are talking for workers in the South Carolina situation, their exposure would be five to ten cigarettes a day...

A. Possibly.

Q. ..in terms of the risk that they are...

A. We are doing a very, very heroic extrapolation, but...all other things being equal, you are right.

Q. So this is the...

A. I say all other things being equal, because

A. (cont'd.) one of the issues that has arisen is that the South Carolina plant is in the center of the tobacco industry, which could be relevant.

Q. Not in terms of the smoking habit of the workers, or just in terms of the asbestos/smoking...

A. The smoking habit of the workers. For example...you see, this graph that we are looking at does not use the smoking habits of the workers. It is saying in this population those are the relativities. In another population, it wouldn't necessarily be the same, and therefore I agree with you in, if you like, the form of the logic you are using, but I wouldn't be absolutely sure that the answer was right.

Q. We are talking to a risk that's from exposure to chrysotile asbestos?

A. Yes.

Q. In terms of the conversion that was made, from...going from your dust counts to fiber counts, how was a fiber defined for that conversion?

A. In what sort of sense?

Q. What is ...

A. A fiber as counted on a membrane filter?

Q. By optical microscope of specific size?

A. Yes, that's right.

Q. Then in terms of...and there are certain limits in terms of the fibers that you can actually pick up and see under the optical micrscope?

A. I'm sure.

Q. And that there's a certain amount of information that is perhaps becoming available because these fibers can also be studied with the electron microscope?

A. Yes.

MR. GIBSON: I would like to enter an exhibit just to help to focus the rest of this line of questioning.

MR. GIBSON: (cont'd.) It's not assuming that you or I have any expertise in experimental toxicology, but I think it may help clarify some of the problems that actually you have been bringing up.

MR. LASKIN: Shall we mark this, Mr. Chairman, which I take it is an extract from the proceedings of this Commission's second public meeting held on December 12, 1980, and I take it as page five out of appendix A, relating to Dr. Chatfield's address.

Exhibit twenty-two, I believe.

EXHIBIT # 22: The abovementioned document was then produced and marked.

MR. GIBSON: Q. Just to put this in context, Dr. Chatfield is the local Ontario Research Foundation expert in electron microscopy for asbestos fibers.

THE WITNESS: A. I do know him, yes.

Q. He was making a point referring to work that had been done by Pott, with animals, looking at a variety of fibers, including artificial fibers, in terms of their potency for causing cancer. What page five shows is a graph of where the maximum effect was noted in animals. It occurs where the length is about twenty microns long, and where the thickness of the fiber is about point one seven five microns.

The problem in terms of measuring asbestos exposure for this effect is that although a fiber may be quite long, that if it's thickness is less than point two microns, you can't resolve it under the microscope. So that it's simply the majority...if you are trying to assess a dust cloud for the amount of asbestos, cancer-causing asbestos fibers, and if this hypothesis is true, the majority of those cancer-causing fibers would simply not show up under the optical microscope.

Q. (cont'd.) Is that your understanding of what...?

A. I have no understanding of the subject, I'm afraid. I don't know. I'm sorry, I don't know, because I don't think it is known. This is part of the trouble. I mean, I don't think we do know what, in human beings, the relative importance of different fiber sizes is.

Again, we are not dealing with one, we are dealing with distributions of fiber sizes.

But if you mean that what we are measuring may be a poor index of the carcinogenicity, I'll agree with you.

Q. Yes, this...

A. And I think we have a beautiful example of this in the problem of looking at...a beautiful potential example in looking at the fiber concentrations in South Carolina, and in Quebec mills. You see, we may really be...they appear to be very different, but perhaps in relation to their carcinogenic potential, they aren't so different. We don't know what we're looking at.

Q. Basically this was what I was about to ask you, in terms of the problem, the anomaly that you brought up this morning, that in terms of what was done with the optical microscope that the dust clouds seem very similar in the Quebec mines and in the South Carolina situation, that perhaps in fact they are not and that, because of the processing, and that explains the greater carcinogenic effect in South Carolina.

A. It's not quite that way around, is it? It is that the dust clouds look very different. They look very different, but the carcinogenic potential doesn't look so different, so presumably the clouds have a different carcinogenic significance, even though they look quite different.

I'm sorry, I'm perhaps not explaining myself

A. (cont'd.) very well.

I mean, the trouble really is, the South Carolina situation would make sense if the dust, if the fiber counts were higher, and the Quebec data would be compatible with South Carolina if the counts were lower. Yet they aren't and one...if we put on one side other possible explanations, then presumably we are not counting the...we are not getting a proper count of the carcinogenic potential of the cloud. They are not comparable, it would appear.

Q. In terms of the straight fiber count?

A. That's right. And it's not, again, so unlikely as it is quite likely that the fibers are quite different in fact...in their dimensions. I don't know if anybody has looked at it.

Q. Asbestos, I believe, is a fairly resistant material, which is the reason that it has a widespread number of uses?

A. Which asbestos?

Q. Both chrysotile and amphibole.

A. The amphibole is a good deal more resistant than the chrysotile, yes.

Q. But even the chrysotile asbestos, in terms of fibers that actually get into the environment and the fair amount of background fibers that you refer to, that we all have a background exposure, it's a common mineral...

A. Yes.

Q. Presumably a fair amount of that chrysotile asbestos is biologically active as far as...?

A. Quite possibly. Probably, probably.

Q. If in fact the processing of chrysotile asbestos makes it, by breaking it down and changing the fiber shape, seems from the South Carolina situation to be increasing the biological activity?

A. Right.

Q. That in fact if the same sort of thing goes on in the environment in terms of road cuts and wind, asbestos, chrysotile that's blowing around, the same thing may be happening, but we don't see that because the background is a very minimal affect anyway, but the form may be more biologically active as it gets messed about in the environment?

A. Yes, or it might be less biologically active. I mean, this is pure speculation as to whether it's more of less. You could think of reasons why it might be more, you can also think of reasons why it might be less. I would guess we don't know the answer.

Q. Coming back to the graph that I presented, in coming back to the problem of mesothelioma and the fact that we don't seem to see much or any mesothelioma that you can definitely tie to chrysotile asbestos, is it possible that this is related to the physical shape of the chrysotile fibers and the way that they are broken down, rather than the actual chemical difference between chrysotile and the amphiboles?

A. I think that would be the orthodox view, that it probably is due to that.

I think that is, if you like, dismissing the chemical differences, which are important. I mean, I don't know if they are biologically important, but they are chemically quite different and so you could, if you were thrown back on it, say yes, but a high iron-containing fiber, if you like, is more important. But it is usually attributed to the shape, and indeed there is experimental evidence to suggest that regardless of what the fiber is made of that if it is a certain size and shape, it will have the same effect. We've got the examples from Turkey, and production of tumors with man-made fibers and so on.

Q. Yes, but that if in fact chrysotile asbestos in small percentage occurs in fibers of the same size and shape as the amphiboles, then it would be...

A. Quite possibly, yes.

Q. There would be a very minimal risk of mesothelioma from it, it wouldn't be detected by your epidemiological methods or surveys at this point?

A. Well, that's what observation has shown, yes. Yes. We are sort of rationalizing it with this explanation, yes.

Q. The mesothelioma, I believe, is a fairly rare condition, as cancers go?

A. It's not so rare in some populations. It's overall rare, yes.

Q. And that a large percentage of mesothelioma has been...can be attributed to an occupational history of asbestos? I believe that's right?

A. That's right. Especially, as I say, where there is a lot of it, where mesothelioma has a high incidence, then a high proportion of the cases appear to be related to occupational exposure.

Q. It seems to be fairly unique in terms of many cancers where there is a variety of risk factors that are associated with a disease, and I'm wondering if it might be reasonable to speculate that the background level of mesothelioma is related actually to the background level of fibers that are actually in the environment of a suitable shape to cause this cancer, rather than some other carcingoenic effect?

A. I think that would be quite high on the candidates for hypothesis, yes. I mean, I don't know any evidence to support it or refute it, but obviously that would be a question you might want to test, yes.

Q. And that presumably a miniscule amount of that might also be related to the chrysotile fibers that are in the environment, but that's without, really, evidence to say one way or the other at this point?

A. I'm not enough of a geologist...I'm not a geologist at all...but I would want to know would be, what is the actual distribution of mineral types which produce airborne particles of the variety which are associated with mesothelioma, before I could answer that. Because it could be that after all there are a lot of other minerals which produce needle-like fibers...a lot. So I wouldn't know the relative importance of asbestos vis a vis the others in this.

Q. Presumably there is a considerable amount of amphibole asbestos that is going into our environment because of the industrial use?

A. And nonindustrial use, yes.

Q. And nonindustrial.

A. Yes.

Q. That we wouldn't expect at this time that that would be showing an effect on nonoccupationally-related mesotheliomas, the rate that we notice in women, although that increase is going on and perhaps the risk is slightly increased, we wouldn't expect that to show up in North American surveys at this time? Is that right?

A. Could you repeat your question, because I lost the thread of whether you meant occupational or nonoccupational.

Q. Nonoccupational. The line of questioning this morning with Mr. Warren was pointing out that the rate in females, nonoccupational situations, seems to have remained completely flat.

A. Fairly, yes.

Q. The point I'm trying to make is that there is some increase in amphibole asbestos going into the environment

Q. (cont'd.) from our use of asbestos, that if the background rate of mesothelioma was due to this background rate of asbestos, we still wouldn't expect that to be showing up as a significant change in the period that you've been surveying for mesothelioma?

A. I'm not sure. You could be right. I just don't know. I don't know what would be the type of pattern I would expect.

I agree that on the face of it there isn't such evidence, that there is any important change and this does tend to suggest that whatever the factor that is responsible for this background level, that the amount of it around hasn't changed very much. I mean, of course, when we say that the amount of mesothelioma in women hasn't changed, this is based on extremely small amounts of data in only one or two places....nearly all that we've collected ourselves.

Q. What I'm saying is that the fact that the rate has remained stable, based on a few cases over a limited period of time...

A. Yes.

Q. ...would not be a reason to throw out the hypothesis that the background rate of mesothelioma was due to the background rate of suitable fibers in the environment, which we assume has gone up a little bit in the last decade because of our use of amphibole asbestos?

A. Possibly.

Q. To return for a moment to the question of gastrointestinal cancer, I believe you found an association between this cancer and high exposures of chrysotile, in your study?

A. Yes.

Q. I think it has come across quite clearly that as far as we can establish anything by epidemiological

Q. (cont'd.) knowledge, cause...that asbestos "causes", in quotes, lung cancer, would be your view, and that as far as, you know, that there was no compelling evidence as far as chrysotile was concerned, around mesothelioma. But somewhere in that spectrum could you place for us how you would see the relationship between asbestos and gastrointestinal cancer?

A. Yes. Well, I mean, I think first of all one has to say that the evidence is much less consistent. That there are studies such as ours in the mines and mills where we have an excess. Our excess overall is slightly higher, for example, than in the insulation workers, though of a similar order.

The exposure-response relationship doesn't lie on a straight line. It is true, as you said, that the highest risk is in the highest dose group, but there is a disturbing lack of straightness about the line.

In South Carolina, there is no excess of lung cancer, even although...

MR. LASKIN: Gastrointestinal cancer.

THE WITNESS: Gastrointestinal cancer, even although it appears to be an area of substantial lung cancer risk. I can't remember whether that applies to Rochdale or not, offhand, but shall we say very roughly half the studies, half the cohort studies show an excess of gastrointestinal cancer, and about half of them don't. And there is no relationship between whether it is the studies with a high risk of something else that have the gastrointestinal cancer, and the ones that don't, don't.

You see, it's an irregular relationship.

So I think an irregularity of that kind is the kind of thing that at once makes you think that the pattern of causation is not simple. It suggests that perhaps there are some other important factors involved.

A. (cont'd.) I do, in a paper somewhere, say that it is almost if we were trying to understand the distribution of lung cancers and asbestos, without knowing that tobacco was a factor. We would have another set of problems.

And it does look...I mean I am now completely speculating, but there is this irregularity, and there it is.

Q. Perhaps at least exposure to chrysotile asbestos has to be considered a risk factor anyway, in terms of gastrointestinal cancer?

A. I think so.

Q. That risk would be experienced, I presume, by ingesting the fiber?

A. It seems most sensible to think that.

Q. And that presumably it is being ingested in drinking water, other fluids, what we eat, it would probably be as effective a way of getting into the gastrointestinal tract as swallowing it in sputum, having...after it had been inhaled through the nose?

A. I don't know, but I would think not. The amount of fiber...any dust...that a person swallows, if he is inhaling, is very large. You must swallow a very great deal more than you inhale, a great deal more. So that I would have thought that the occupational-exposed person would be quite heavily exposed in the gastrointestinal tract.

I don't know what you think. I think that.

Q. Yes. Granted that...

A. By swallowing the material that he sort of coughs up, or whatever.

Q. Granted that the person in the occupational situation would have an exposure several orders of magnitude greater than anything that one might swallow from exposure, environmental exposure, whether airborne chrysotile fibers or in the water?

A. Yes.

Q. But presuming when you actually take...if it's in the gut, presumably, the biological activity would be relatively the same however it got there? It would be just, it would be a difference of exposure that we were talking about?

DR. UFFEN: Does that mean that you can get exposed to the same fiber a couple of times, once in your lungs and once when you swallow your spit?

THE WITNESS: No.

DR. UFFEN: No?

THE WITNESS: I think the one fiber can only finish in one place. I think it either stays in your lung and does the damage, or you clear it from your lung and swallow it, or conceivably spit it out.

DR. UFFEN: It couldn't do any damage on the way through? Only where it ends up?

THE WITNESS: That would be a bit farfetched.

DR. UFFEN: How would you know...?

THE WITNESS: I don't know what damage it could conceivably do on the way through.

DR. UFFEN: Well, I think of little needles working their way through my lungs and my intestines, I kind of wondered if they don't do any damage.

MR. GIBSON: Q. I believe we were talking also this morning about the asbestos-cement industry, and that the experience of the workers in that situation did not seem to be dramatically different to miners and millers?

THE WITNESS: A. That's my impression, yes.

Q. I also believe that a large amount of asbestos cement is used for pipe?

A. Yes.

Q. And that pipe is used for carrying drinking water?

A. That's right.

Q. I believe it's also possible for fibers to leach out of the pipe and into the drinking water?

A. I'm told it is. Especially before it gets coated. I believe after the water has run for awhile, it doesn't.

Q. That...would it be a simple epidemiological matter if you were trying to look at the exposure of large cities to gastrointestinal exposure to chrysotile fiber because it was coming into their drinking water from pipes...well, let me put it this way. It doesn't strike...because of the many other factors involved in gastrointestinal cancer, that simply taking two cities of eight million, and if ten GI cancers in one of those cities was due to the asbestos pipe, because of all the other factors you would never be able to tease that out by epidemiological methods?

A. No.

Q. That in terms of this sort of risk, we have to play around a bit with the biological possibility that something is or isn't happeing?

THE FOREGOING HAS BEEN PREPARED
FROM THE TAPED RECORDINGS
OF THE INQUIRY PROCEEDINGS

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A. I should think we come back to this other business, that I don't see how you guess what the effects of extremely low levels are, except by extrapolating from the higher levels; and that means accepting some form of model, a model which is an act of faith. I don't know any other approach to that question.

Q. But if we take ---

A. Oh; excuse me. May I say, yes, I do think -- I think, in the longer run, it is conceivable that we could make progress in this by the wider use of the electron microscope in large-scale surveys. But nobody's begun to do this.

Q. But taking the model that you've developed, where there's a finite risk all the way down to zero, and presumably, if that applies to chrysotile as a risk factor for gastrointestinal cancer, and that there's a small exposure being created through the use of asbestos water pipe, that there would be some excess cancers related to that exposure, although very few. But, following from the model that you've developed, we have to make that assumption.

A. I wouldn't make the assumption, I'm afraid. I'm following you entirely in your line of argument as a possibility; you know, as a speculative hypothesis.

I feel that we would be rather wrong to say, it looks as if that must happen. For example, I mean, I think we have to say straight away that we haven't got this kind of encouraging consistent evidence of cause and effect in gastrointestinal cancers. If we had, I'd be a little bit more happy. We don't have it; so it looks as if almost certainly there are other factors involved.

If there are other factors involved, I don't know what those other factors are. For example, it could be that asbestos had nothing whatever to do with gastrointestinal

A. (cont'd.) cancer, and I'd like to give you an example.

There has been observed for some time an association between calcification, calcified areas in the pleura in asbestos workers, but the evidence is now building up that it has nothing whatever to do with the asbestos but, rather, connected with another mineral which is very closely associated with asbestos.

We've just been investigating an epidemic in which sixty per cent of the adult population of a village in Greece have got severe calcification, and there is no asbestos around.

In Thetford Mines, something like ten per cent of the older workers have calcification; in Asbestos, sixty miles away, it's almost zero.

So what I -- I'm only giving you that as an example of what you might easily say, here is a manifestation which is probably due to asbestos; and then -- which is all right, for practical purposes. It might be -- as long as we don't want to worry too much about whether it's true, as a sort of rough-and-ready marker, it's useful.

But if you then want to extrapolate from that argument and say, yes, it is due to asbestos and it is the asbestos that leaches out from the pipe, and therefore we will get asbestos-related troubles from the water, I think you're jumping a few steps, such as that it mightn't be the asbestos and that the factor responsible doesn't leach out. For all I know, it may not be even in the pipe.

Do you follow me? I feel, what you're saying is entirely a plausible possibility, but I don't think we're at the stage of being able to take it as a probability; not in my view.

Q. That's what I wanted to establish; if it was a possibility that simply -- that had to be -- you'd have to consider, even though there are great deaths in our knowledge at the moment.

A. Yes.

Q. I believe, also, that you said that the levels of exposure in the Quebec mines have come down to a level that -- and in terms of the risk to miners and millers, that this seems to be acceptable to the workers. It may well be that they would say that, in that specific situation, it would be a risk that they would accept.

A. I couldn't tell you whether they think it is an acceptable risk, for certain. What I would say is that it has dropped very strikingly, and, therefore, it ought to be more acceptable than it was. But whether we're now at a level which is acceptable, I can't say.

Q. Let us assume, actually, for the moment that it is at a level that is acceptable; we've made that assumption. I'd like to just explore, for the purposes of this Commission, that the consequences of taking a ton of that chrysotile out of the mine in Quebec -- or Russia or Italy, you know; I don't mean to ---

MR. CASGRAIN: Discriminate.

MR. GIBSON: Q. --- discriminate -- and that there are other costs -- I mean, that the particular cost to the individual worker and the mine has been accepted; but in terms of the actual -- all the costs that the Commission has to consider, that some of those workers, over a long period of time, will develop some health-related problems that will generate some costs for society, presumably, that also have to be considered.

A. You mean in the use of this ton of stuff?

Q. No -- well, just in terms of the workers who take the ton of the stuff out of the ground and mill it.

A. They have a certain ---

Q. They're going to experience a certain amount of morbidity because of that process, which they have accepted, but it's still going to generate a cost for society in terms of their health care.

Something is going to be done with that ton of asbestos. I presume that it's going to be put into processes that may increase its potential for causing cancer, depending on the use that it's put; that there are workers in that situation that are going to be exposed, who also may have to accept some risk.

If one of those uses is, for example, brake linings, besides the workers who do the primary processing, there may be people like mechanics who are repairing brakes, who also enter into the picture, and again are exposed to that asbestos; and further changes may have taken place that increase or decrease the risk of cancer, but that it's still there.

And I presume, ultimately, that this asbestos is going to end up somewhere in the environment, whether it's floating around in our city air, from its use in brakes, or in the water pipe, and that it will be around for a fairly long period of time.

And that, at least, we cannot assume that, simply because the worker exposure is over, that there's no hazard from it, now that we've actually finished with the stuff, and done with it, and it's sitting out there in the environment.

Am I correct in all those sort of evaluation of the costs of chrysotile asbestos?

A. Are you prepared to consider anything on the other side of the balance?

Q. Certainly.

A. I mean, I just wonder, because, obviously, in any cost benefit, you've got to consider all the harms that are possible -- I mean, we milk cows and we kill people with the milk, and we make margarine and we kill them with that, et cetera. You know, we don't do it just to kill people; we do it because we want the stuff, and we want it for purposes which society, rightly or wrongly, thinks it wants. It wants motor-cars to stop.

So we have to balance the cost of the possible risk of a cancer somewhere against a child that doesn't get run over by the car, et cetera. Now, I don't know how you balance those things, but what I don't find any difficulty -- much less difficulty -- I mean, if you were to be saying -- producing the same argument in relation to, should we grow tobacco, I would have much more difficulty in defending the social values. There are lots of things -- or alcohol, or a whole lot of things which society says it wants, and where the benefits are less certain.

It isn't for me to talk about this; I have no expertise in it. But I do feel that what you're saying is a very sort of incomplete ... I can't agree with this as a picture of what we've got to deal with; that's all.

I can agree with it if you say, "I am trying to look for every possible adverse point against asbestos." I would say you haven't left out much, and I would then agree with you. But I don't think that that would imply -- I wouldn't want to agree with that as an overall balanced view of the problem we're trying to deal with.

If it were as simple as that, we'd obviously get rid of it, wouldn't we?

Q. I only have a couple more questions based on

Q. (cont'd.) that; that is someone came along with a magic wand and could give us a safe substitute for chrysotile asbestos, would you feel, in light of what you know about the health risks from chrysotile asbestos, that it would be wise to use that substitute?

A. I'd certainly want to be assured that it wasn't of the same size and shape.

Q. Yes; I mean that's ---

A. You know, in view of the fact that the asbestos is used because of its size and shape, we run into a problem, you see. I mean, of course you're right; if you can get the same effect with something that is square, or something, fine; you know, I'm sure this is a very good idea.

Q. Looking at your own experience, not as sort of a matter of -- from the point of view that the Commission has to consider this, or governments have to consider this, but from your sort of experience of looking at the risks of this substance as compared to other substances, and the epidemiological effort involved, and adding up these various points at which asbestos creates a problem, do you think it would be worth the effort for society to try to determine safe substitutes, or alternative ways of -- completely alternative technologies to do the same task in a different way that you don't need something that has the same size and shape as that fibre?

A. I bet you that industry's working very hard at just that. Yes, of course it is worth doing, particularly if you can sell the product. Yes, of course it would be a good idea.

But I still think there are other sort of steps along the road which are less radical. I mean, one is, I would have thought that society knows enough now to say we will not use amphiboles.

A. (cont'd.) I would have thought society knows enough now to say we will reserve asbestos, even if we hate it, for certain uses where it is socially very desirable.

And the full extent of that social desirability -- and I've got no shares in the stuff -- I think really has to be appreciated, not in this continent but in, say, Africa.

All that we're talking about is just superficial, minor hazards compared with the problems of a world which doesn't have water supplies; water supplies which they will not have if they don't have asbestos pipe.

I only bring that up as one little point. I think we're taking an extremely privileged and extremely self-centred, selfish view of this whole problem. That doesn't mean we shouldn't look after ourselves; I'm sure we should.

But I think that we should take a view of what this stuff's for. I wouldn't put anything into trying to have no motorcars, but you won't have motorcars for some years to come if we say we're not going to have any asbestos. Or, if we do, they'll be very dangerous motorcars.

By all means, I'm sure industry will try to, you know, develop safer substitutes. I just hope that when they develop those safer substitutes, they will take into account that nobody knew the hazards of asbestos until they'd been using it for about seventy years. So we have to be a wee bit cautious.

And I do say that particularly in relation to, you know -- the magic wand can't change the fact that one of the reasons that asbestos is useful is because of its shape and because it will bind. So you really want to have a magic wand which can do the same thing without doing that.

Of course, if society can do it, fine; and I'm sure they should. It's a rather ---

MR. GIBSON: I have no further questions. I appreciate the concern that you've raised. I hope that its use in other areas is for -- in places like Africa -- is for socially desirable purposes.

THE WITNESS: I think water's quite socially desirable.

MR. GIBSON: Thank you, Dr. McDonald.

DR. DUPRE: Mr. Starkman, are you next?

MR. STARKMAN: Yes; thank you.

CROSS-EXAMINATION BY MR. STARKMAN:

MR. STARKMAN: Dr. McDonald, with previous people you've had an opportunity to discuss the effect of asbestos on the incidence of heart disease, and now gastrointestinal cancer, and I would like to have your comments on the effect of asbestos exposure on bronchitis and other air-passage diseases, and to know whether you found -- what incidence of that you might have found in your cohort study.

THE WITNESS: Well, we didn't find anything very convincing in the cohort study; but in our studies of current miners, we did make studies, particularly using questionnaires on coughs and breathlessness on exercise, and so on.

And, in that, there was I think fairly strong evidence that the bronchitic disease, the productive cough, was related to dust exposure.

Now, I think what we haven't followed up on, and, indeed, I think it is quite possible that the this bronchitis which, after all, probably does occur in other dust-exposed workers (coal, and so on) -- but in the production of the bronchitic reaction, it could well be that the serpentine dust is quite as important as the asbestos fibre. You know, I mean, this is one of these situations where we really don't know. We're looking at dust.

THE WITNESS: (cont'd.) Again, I think that if you go down to the -- I would leave it there; I can't say any more. I don't know whether, shall we say, textile workers have this same evidence of bronchitis that the miners do.

MR. STARKMAN: Q. Certainly, in your opinion, exposure to asbestos fibres contributes to bronchial conditions?

THE WITNESS: A. I should think it probably does.

Q. And to take that further, what about that bronchial condition as a cause of death -- as a cause of death or contributing to death?

A. I think a good deal less important than the kind of diseases we've been talking about. But, I mean, I would think that it will make a contribution, yes.

But I repeat again: I think that this could well be a function of the dust, the total dust, as a factor, rather than the -- this is one of the situations where I'm not so sure that measuring fibres is as relevant as total dust. And I say that without knowing whether that's true or not.

Q. There's been no work to determine, out of a dust cloud, whether it's the asbestos dust as opposed to any other dust which is causing the bronchial condition; is that what you're ---

A. This is right. This -- perhaps you could say this; but we've recently been conducting a survey, a radiological survey, of miners in the eastern townships of Quebec, men who have been exposed to much lower levels in the past -- these are now not adequate, but much lower levels.

And it's very interesting that the actual pattern of the X-ray changes is different; whereas, before, we used to see the irregular opacities and all that is typical of

A. (cont'd) asbestosis. We don't see this so much now; we're now beginning to see a sort of fine punctate type of pattern, such as you see in very early coalminers' disease. And that makes me wonder whether we're now not seeing relatively more of what you might call the non-specific-dust effect.

And that doesn't mean disease, of course, just because you look at little opacities; as in coal workers, the opacities are simply -- you're looking at dust; you're not looking at disease. But it could be that this is in line with what I'm speculating.

Q. Other than the diseases that we've already touched upon (gastrointestinal cancer, perhaps bronchitis), are there any other illnesses which you feel can be traced directly to exposure to asbestos dust or particles?

A. Not in a specific way. I expect you know that there has been speculation about laryngeal cancer and asbestos; we haven't found that. I mean, shall we say, some studies have found an excess; some haven't. It remains not a very numerically important disease, although it's not nice to have.

And I think, otherwise, I would think that most of the other effects are probably of the kind we were discussing earlier; that is, the overflow effect of a disabling disease upon almost whatever you have.

I mean, it's interesting, even cardio... -- I'm sorry; vascular disease of the nervous system shows an increase in heavily exposed people. Well, I mean, there's no way in which -- no rational way in which you could imagine asbestos dust affecting the arteries of the brain; but the fact that they have apparently a higher rate, I would have thought, was simply that if you've got a bit of asbestosis, too, your survival chances are less good.

A. (cont'd.) So what I'm trying to say is, no, I don't think there are any other specific diseases. There's been speculation on several, but I don't know of any strong evidence.

Q. I wanted to just dwell for a moment on the shape of the dose-response curve, because, as I understand it, you said earlier that your data, at low doses -- your data is compatible with a number of parameters for the dose-response curve, one of them being lineal parabolic, and, I guess, any number of other possible combinations.

I was just wondering why that's so, you would conclude, or feel most comfortable (if I can put it that way) with finding that a lineal relationship is probably the better one, if I could put it that way.

A. Well, first of all, my statistical colleagues would, I think, say more strongly than I do that it is -- it points to a linear relationship. In other words, when I say a thing is compatible with others, it's taking a rather more kind of tolerant view of the observations, and saying you could fit them into other shapes; and you could.

But what we observe fits very well into a straight line, at the lower end. I mean, it isn't reasonable to think that it will go on and on forever. I mean, we don't look to see, is it straight forever; presumably, it eventually flattens out.

Q. At the upper end?

A. Presumably. So why do we pick it? I mean, I think, again, because our cancer colleagues say, "That's what we'd expect," you see. Now, perhaps not all cancer colleagues will do that. It fits at least one group of people's concepts of probable mechanism.

What other reason? It's a very useful model; it

A. (cont'd.) has the attractions of being very usable.

Q. In your professional opinion, is it the one you'd feel most comfortable with?

A. Yes. I think it probably -- it could be said to have an error either way at the bottom end, but, on balance, I think, in relation to most alternative views, it is a safer view.

Q. Right. And we're talking about lineal for mesothelioma and lung cancer?

A. I don't think we know about mesothelioma; we haven't got -- we haven't, certainly -- but I don't think anybody has any adequate information to really tell you what the model is in mesothelioma.

Q. Can I just look with you at tab 17, on page 7.

A. Yes.

Q. And I'm looking there at figure 3.

A. Mmm-hmm.

Q. And I guess I'm just asking for some clarification of this diagram, because the way I understand it, it is that, for chrysolite [sic] miners and millers, the latency period for mesothelioma seems to be -- and granted there aren't all that many there, but what evidence there is, they seem to be concentrated in the forty-five to fifty-five years from first exposure.

And, to me, that indicates that, in fact, there may be a lot of mesotheliomas still to come, because, if we take the mean of, let's say, fifty there and go back fifty years, that means we're looking at someone who had first exposure in 1930, which is, again, in a time before we had measurements and when everyone acknowledges that the dust counts were very, very

Q. (cont'd) high.

So, somebody who had their first exposure in 1950 wouldn't get -- wouldn't have mesothelioma until the year 2000, assuming that this gives us an accurate reflection of the latency period.

A. Well, of course, it doesn't. What you are sort of saying, I think, would be supported if most of these cases, if you like, were old people, and therefore we're going to see the further cases when the younger ones get older; well, they're not, you see. I mean, that's the first point; they are scattered right across the age range and in relation to different times of starting work.

It is, of course, possible that, as only half the cohort has died so far, that we will see more. In fact, I think in this article we say we will probably see more.

A point to bear in mind, however, is that, today -- I mean, this is up till 1975 -- the age of the cohort, the oldest one is - what is it?

MR. LASKIN: Ninety.

THE WITNESS: Ninety; and the youngest one, fifty.

So the cases are going to be -- tend to be quite old from now on. And I'm not saying that a person who dies of mesothelioma at ninety is less important than one who dies at forty; but I think in most people's mind it would be.

MR. STARKMAN: Except to the person who's dying.

THE WITNESS: Even in the person's mind, frankly. Frankly, I think that the concept -- this hasn't come up, incidentally, in our discussions.

One of the marked limitations of all the analysis we've been doing is that it gives equal importance to a death at ninety or at forty or at twenty. And, I think, in social terms, that is a pity.

THE WITNESS: (cont'd.) However, I mean, I'm not disagreeing with you. I don't know how many more cases there'll be. Half of them died; I would have thought it's very unlikely that it'd be more than half -- more than double that, but could be.

The thing, however, that I should draw attention to here is that this shows the interval from first exposure in the chrysotile industry to death from mesothelioma; and what we notice is a very wide range, in contrast to the very tight range for the crocidolite cases; crocidolite cases in which the etiology, I think, must be fairly clear-cut.

To me, what this suggests, one, it may be that the chrysotile-producing effect is much weaker, and that, for some reason, we have a much longer latency, or it could be in line with what I was saying earlier: that it's nothing to do with the chrysotile.

But that, in the course of a working lifetime, the longer you have, the longer you have to be possibly exposed to something else, even in the chrysotile mining industry. They use crocidolite in Asbestos; you know, there's a factory which uses it, and so on. And there is tremolite in the asbestos to which they're exposed.

So I think there is room within this for the role of other factors than chrysotile; indeed, I think it almost points to it. But that is only just speculation.

MR. STARKMAN: Q. Can I -- in doing your study of your cohort, I take it you visited Thetford Mines and Asbestos on a number of occasions?

Yes?

THE WITNESS: A. I can't count them; yes.

Q. Could you give the Commission an idea of what the population would be of these towns?

A. The current population -- somebody can correct me -- it was of the order of twenty thousand in Thetford Mines and around ten thousand in Asbestos.

Lesage is nodding, so I expect that's right.

Q. And, of that, what percentage, roughly, would be working in the asbestos mining and milling?

A. The current work force in mining and milling is around six thousand, of which rather more than two thousand are in Asbestos and about four thousand in Thetford; so, what shall we say -- that a third of the population are males of working age. Is that reasonable? I don't know.

So what we've really got is a similar proportion, let us say, two thousand out of ten thousand -- they're all men, so that's two thousand out of five thousand; then there are the children and the old people, so it probably must be over half the workers.

Q. And I just wanted to deal with one more aspect of this death certificate issue.

MR. CASGRAIN: Sorry; you're referring to Asbestos now?

THE WITNESS: That was Asbestos. In Thetford, where we've got twice as many workers ---

MR. CASGRAIN: But you have a far larger population of workers for other employments as well.

THE WITNESS: That's, I must say, what I would have thought, but, nevertheless, the City of Thetford is twice as big and the number of workers is twice as many.

But, in Thetford, it is true that the workers come in from surrounding villages more than they do in ---

MR. STARKMAN: I'm happy to leave it go at just some rather gross numbers. I was really asking the question because I wanted to just get at one more aspect of this death

MR. STARKMAN: (cont'd) certificate issue, which is that, I take it, in the Town of Asbestos, the suggestion was that doctors, who were working in an asbestos area, would more easily identify, or more readily identify, deaths as a result of asbestos than in areas where they wouldn't necessarily be looking for it.

Q. Say, in the Town of Asbestos, do you have any idea of how many medical practitioners there would be?

THE WITNESS: A. I'll be guessing, but I'll guess ten. How many would you expect?

I think about that; ten to twelve.

Q. And do the workers there have annual checkups?

A. In the -- you mean, in the ---

Q. In the industry.

A. --- the occupational health department; yes.

Q. Excuse me; what did you mean, in the occupational health department?

A. Well, the industry has an occupational medical service, and they, for something like -- since 1935, have had a quite good occupational health service; and that is quite separate from the -- they don't provide medical care in the ordinary way; that's provided by physicians in the community.

But there's the occupational medical department, with two or three physicians who work there.

Q. And that's a company clinic?

A. That's right.

Q. And we had some mention that, when people die and their death certificate is filled in, would we have any knowledge as to whether these people died in that area; in other words, near Asbestos, or in the eastern townships, or might they have died in Montreal or in some other place? Do we know the place of death?

A. Yes. I couldn't give you an immediate answer. I would guess that about half our cohort died in the eastern townships; not necessarily in the same town. Quite a lot of them have migrated, on retirement, into bigger places, like Sherbrooke, into Montreal, into Quebec City, Victoriaville, so on. There's a certain tendency towards -- older people to go out of the country into the bigger towns.

And I was told, when I first undertook this study, that the beauty of it is they'll all be still in Asbestos and Thetford; well, I can promise you, they're in every country of the world. So they go everywhere.

Q. The spin-off to my question is that, even if you assume that doctors working in the area are more likely to recognize death by asbestos, half of the cohort didn't die in the eastern townships; they died in other places?

A. But half did.

Q. Yes.

Can I look, just for a minute, with you at tab 18, on page 14, and I'm just looking at the beginning of the first full paragraph in the lefthand column -- tab 18, page 14.

A. 18; the first -- can you repeat the ...

Q. Yes; the first full paragraph on the lefthand side.

A. Yes.

Q. And it says:

"Relative risks of lung cancer were considered in detail by Liddell et al, and it appeared that there was little to suggest that the way in which dust exposure had been accumulated played any part in determining the risk."

And I guess my question is, is that a reference to what was discussed before, about peak periods of exposure and

Q. (cont'd.) intermittent exposure, or is this referring to some other method of dust accumulation?

A. No; it is -- I think the peak exposure concept is a little different. I think the peak exposure concept is that you may have a prevailing level which may vary, but that only now and then, say, one of the crushers, or something, one of the things will break down, and somebody will get not just, you know, shall we say, the ten fibres but a whole waft sucked in his face of very high concentration, and that might last, shall we say, ten minutes.

The way I've interpreted this discussion of these sort of excessive peaks is in that sort of way; of a very sudden -- of a sudden, rather overwhelming high amount.

What this deals with, there is no way we have had of even looking at that sort of phenomenon. But, as against that, we have other -- another kind of issue.

For example, a man could work -- his life history might be composed -- what it won't be composed of is working at about the same level for all forty years. It may be composed of a life in which he was heavily exposed in the earlier part of his life, and then much more low -- a low exposure -- or he might have had a job which gave him relatively little exposure and, in the last twenty years, it was high.

So both those patterns of exposure, which are diametrically opposite, would, on our index, add up at the same thing, essentially. And what was done here, and what this refers to is some analyses which our statisticians did, which was to look at different patterns of exposure; in other words, to what extent did the average stay really steady, or was it high at the beginning or high at the end?

And, in relation to that, it didn't appear that the pattern had very much effect. But it doesn't have any

A. (cont'd.) bearing on this peak exposure question.

Q. Have you ever made that calculation for smoking? I'm talking here about the interaction between smoking and emphysema.

A. Funnily enough, nobody has even for cigarettes and lung cancer, you know, that I know of. It's one of the interesting things that we've been discussing exposure very critically here, but we haven't discussed exposure very critically in relation to a very well-accepted phenomenon like lung cancer and smoking, where you ask somebody, "Do you smoke or not?" and "How many cigarettes do you smoke?" and, lo and behold, you get a linear relationship.

But I don't think anybody has looked at this sort of thing. Besides, I don't think people vary very much, you know. I mean, unfortunately, people mainly start smoking and then keep on smoking.

Q. I was thinking about the possibility that they might stop smoking.

A. That is possible, but not very frequent, unfortunately.

Q. I wanted to ask you; at the very beginning of yesterday, you mentioned the fact -- you mentioned selectivity in hiring, or selectivity in the work force, and then we had some conversation about the -- particularly, the good health, or relatively good health, of mining workers, mining/milling workers, but I thought you were talking about something even beyond that when you mentioned it; that there was something that would select out mining -- asbestos mining and milling workers as opposed to other mining and milling workers.

Are there any criteria --

A. Oh, no; I wasn't referring to that. I meant

A. (cont'd.) that in any community, people apply for the jobs that are available, and they're selected or not, according to whether they, you know -- the sort of job market and pressure, and whether they meet the physical and other requirements for the job. So there will be a selection process, and part of it is, I suppose, physical.

Well, not so much selection, because I don't think there's been very much medical selection of workers in the asbestos industry. But, if you're sick, you don't go for a heavy job.

Q. Or at least you won't be hired for it.

A. You might be, but you won't go for it; I don't know. I was saying there wasn't a lot of medical screening out in the earlier days. It used to be very casual.

Q. The last area of questioning I wanted to touch on was the question of, when there is some fibrosis detected in an X-ray, or deduced from a lung function test, and it's suspected that it may have been caused by exposure to asbestos fibres, what, if anything, is the medical treatment for that condition?

A. Of asbestosis?

Q. Of asbestosis.

Well, I'm talking about at first detection. If you caught it at earliest detection, and perhaps you might comment on what would happen ---

A. I'm not aware of any medical treatment, in the ordinary sense of the word "medical treatment," which would have any therapeutic effect.

Q. I guess the thrust of my question is, you said, well, it may not -- if someone was diagnosed as being asbestotic, it may not matter whether or not they continued to work.

In that determination, you know, there must -- it

Q. (cont'd.) seems to me there must be some cut-off point, where, if you're saying this worker is asbestotic, I don't think further exposure is going to matter but, in the initial stages, wouldn't it make a great deal of difference whether they've got additional exposure?

A. Oh, yes; I mean, that is the issue. In other words, the importance of early detection is not so that you can say we can allow the dust level to go on at as high as you like, but we'll take this man out of it.

What I think is important is that you detect it and say, that must mean that our dust levels are not satisfactory and that, therefore, you should -- the moral is, to put the dust conditions right so that it would be perfectly all right for him to stay in the job.

But, over and above that, beyond a certain age, I think we could say that it will make no significant difference to his, if you like, expectation of life, progression of disease, because it's too late to make that difference. By the time a man is, shall we say, sixty, it will not make a difference.

Q. But if fibrosis is detected, let's say, in a man's twenties, then it would make a difference?

A. It wouldn't make any difference to the disease that he has to take him out; it would make a difference to whether he were to be exposed to further dangerous levels of dust. Do you follow me?

Q. Yes.

A. Making a difference. There is no effect on the disease by taking him out. It's simply that you're using this as an opportunity to save him from further exposure.

But the other way of saving him from further exposure is to protect him.

Q. But in this context, is there any evidence to suggest that some people are more susceptible or likely to develop fibrosis, and that that fibrosis is more likely to become asbestotic?

And I say that because we were talking before about the context of overwhelming defence mechanisms, and it occurs to me that different people would have different defence mechanisms for specific ---

A. There is a lot of speculation on this subject, and there has been a certain amount of looking for evidence of people who have an abnormal or a different type of immune mechanism; because there is other evidence from, for example, coal workers that susceptibility to fibrosis in coal is, to some extent, affected by the inborn characteristic in this respect to the worker.

It is true, I think, as a generalization, that there is no exposure to anything to which men are uniformly susceptible. Susceptibility varies to any factor.

So, undoubtedly, there must be people who vary in their susceptibility.

In occupational medicine, you may be aware that this is a phenomenon that, in general, people try to avoid thinking about, because, in particular, it has always been the argument that it could be said that it is not the conditions that are bad, but that the workers who suffer are unduly susceptible; okay.

And I can well see that, for practical reasons, that is a very bad argument to follow. It's much better to say, we want conditions in which even the susceptible can work. There are ...

You can take that a bit far sometimes, because, I mean, for example, you wouldn't allow a man who is smoking

A. (cont'd.) heavily to work with gasoline, but, so far, you are prepared to let him work with asbestos, although it is very dangerous for him.

However, apart from advising him, again, it is considered his privilege to work; so, yes, susceptibility will vary. But I do think that this issue may become an important issue, if we manage to get levels to a very low level.

Because it may be that, if you like, the last five or ten per cent may be very difficult to protect. You see, I'm not just thinking about asbestos, but, in all forms of environmentally produced disease, to reduce environmental -- to improve environmental conditions to a very good level entails a lot of work.

If you like, the last five per cent is extremely difficult and extremely expensive to get it really down to zero; and that's the point at which -- you're doing that in order to protect, probably, the small proportion of people who are very difficult to protect.

And, therefore, I think we may move to a situation in which looking for evidence of susceptibility, if it were possible to identify it, could become practical -- a practical issue. We're not at that yet.

I don't think there is any way, at the present time, of identifying the man who is at special risk from getting fibrosis.

Q. I wanted to just ask you -- I know you haven't written much on this, but it's on the question of compensation, and I guess I'll ask you, as a medical doctor, whether you've had any experience with diagnosing these cases.

Obviously, the cases we're talking about are the ones that are more difficult to diagnose, because we can either agree or disagree; there never seems to be ---

A. I've had no -- I've never worked on a compensation board; I've never had any such job. I have no experience of that.

I've got some thoughts on what you might call the epidemiological background of compensation.

Q. What do you mean, the epidemiological background of compensation?

A. Well, I mean -- I presume that compensation is social adjustment for the effects -- for a cause-and-effect relationship. In other words, we're saying that this is the cause of something, and, therefore, we're going to compensate him for having suffered from that cause. And as cause and effect is an epidemiological issue, yes.

So, I mean, one can, of course, see, if you like, the problems; in other words, there isn't any serious problem in -- at least, I don't see any insuperable problem in identifying levels of disability and of levels of asbestosis, for practical purposes, in people who have been -- worked with asbestos.

And it seems to me that one can probably fairly readily -- this is obviously not a readily solved subject, but there are ways in which you can make an assessment of the disability and the attributability in asbestosis.

At the other end of the scale, on mesothelioma, within certain limitations, it seems to me, that while we -- and I would strongly emphasize again that not all mesotheliomas are due to asbestos, but such a very high proportion are, and it is so difficult to prove that they aren't, that I would have said, for practical purposes, a mesothelioma case, that has a history of work compatible with it being caused, again is a fairly straightforward issue.

But what I think is a very unstraightforward issue is lung cancer or gastrointestinal cancer.

Q. I believe you just said that it would be a fairly straightforward matter to determine the extent of disability and attributability to asbestosis. Would you just do that off reading the X-rays and taking the lung-function tests and their departures from normal; that would be the extent of the disability. Is that how you'd make that determination?

A. I'm not in this, but if I were a member of a medical panel, I would try to assess his degree of disability from not just function tests, but function tests, X-rays, history, and so on.

Q. Did you ever look at your cohort to see how many of those people had been receiving compensation for any ---

A. No, no.

Q. Would that information be available to you? I guess it would be.

A. It conceivably would; I'm not sure.

MR. STARKMAN: I have no further questions. Thank you, doctor.

DR. DUPRE: Mr. Ublansky?

MR. UBLANSKY: It's already been covered.

DR. DUPRE: Dr. Uffen.

DR. UFFEN: I have sort of a tidying-up question.

If you don't mind going back to the dose-response curves again, your paper, at tab 18, has been referred to over and over again, and then the later on, at tab 21.

They both have graphs in them of standard mortality -- one's SMR versus dose, and the other is relative risk.

In those cases -- and I want to look at both ends of the straight line -- does the straight line go through the origin because it just happened to go that way or was it forced through the origin as a starting point?

THE WITNESS: Well, on page 19, the spots are

THE WITNESS: (cont'd.) where they -- are observed spots. The line was fitted by a modified least-squares technique, which is sheer black magic to me; but I presume that is the statistical technique for fitting a line to the observer.

DR. UFFEN: But, normally, that would mean, you don't put the origin in as one of the data points?

THE WITNESS: Oh, no, no, no, no; definitely not, no.

DR. UFFEN: What about the later graph, relative risk; that's tab 21, page 813; now, you and your colleagues got a more precise ---

THE WITNESS: Relative risk will go through the origin, by definition.

DR. UFFEN: By definition; right. Thanks.

Now, at the other end of these graphs, when you're getting out to the heavy doses, by the time you'd got to the report at tab 21, you'd accumulated more information; and that last point, way over to the right, is pretty close to three thousand millions of particle per cubic foot; and is that over a forty-five-year lifetime?

THE WITNESS: That's over whatever lifetime it was, I think. You see, this is based upon the actual cases and how many particles years they'd actually accumulated.

A lot of these people had worked well over forty years -- fifty years; even up to sixty.

DR. UFFEN: I tried to make a -- I don't know whether I've done it correctly, but I've tried to make a little calculation of what sort of exposure corresponded to that last point out there, and I got a kind of what seemed to me to be a huge one, and it seemed to be over a hundred ---

THE WITNESS: Particles.

DR. UFFEN: --- particles -- hundred million.

THE WITNESS: Yes; it's almost unbelievable, isn't it? It's quite possible.

DR. UFFEN: Where would they be getting exposed to that now?

THE WITNESS: Oh; not now.

DR. UFFEN: Not now.

THE WITNESS: Thank God; no. These are people who -- I wonder, can I show you -- you see the average for the industry in 1950 -- the average for the industry -- was seventy-five million particles.

DR. UFFEN: Then you must have had even huger ---

THE WITNESS: In the worst mills, it was well over a hundred -- well over.

DR. UFFEN: Well, how come these people came to light between 1966 and 1975?

THE WITNESS: Well, I mean, they died in that time. These are the members of the cohort who had been traced from way back, and they died; and then we looked back -- do you get me?

DR. UFFEN: I see now.

THE WITNESS: But I must agree with you that these exposures are extremely high. And I can remember Dr. Nicholson, who you'll be seeing, I think, next week, saying, "I don't believe that that amount of stuff can stay in the air." But, as a matter of fact, I don't think it can.

I've seen the workers where you couldn't see the workers; I mean, literally couldn't see them. They were in a cloud that you couldn't measure. I mean, I'm not speaking of throughout, but I'm speaking particularly of the bagging department, where it was sometimes a habit of even getting into the bag and jumping on it, to pack the stuff in.

Now, I'm not speaking of what they do nowadays, or

THE WITNESS: (cont'd.) not even in the last twenty years; but in the 1930's and the twenties (some of these people would have been working even then) this was quite possible.

DR. DUPRE: Dr. Mustard.

DR. MUSTARD: Dr. McDonald, your tab 18 is an interesting document, and I realize it's simple to take a look at all that lovely data displayed out in that manner and that one really probably can't draw much conclusions from eyeballing some of the data in tab 18.

But, as you know, you've looked at many comparisons within such a table, and at least raised some questions. And one of the questions I have is, if you look at laryngeal cancer -- page 18 and 19, yes; it goes through the whole business -- if you look at it, the incidence of laryngeal cancer in table 7(b) is about .21 per cent of the total population of people you looked at, if I add up all the cases -- and my calculations may be wrong, because I didn't have a calculator, and doing it in my head's dangerous -- but, in essence, in 7(b) and 7(c), laryngeal cancer, as a proportion of the total cohort in that sector, is about twice that in 7(b) -- sorry; 7(a) and 7(b), it's twice that what it is, approximately, in 7(c) and 7(d), which is rather interesting, because I would have thought it would have been the other way around; that the longer exposure, et cetera, it would have been a greater risk.

But the question that came to mind is that, I suppose if laryngeal cancer can be caused by asbestos, you have to inhale the particles in such a way that you can dump them in the larynx; it's a little bit different than taking them in in another way. And I suppose, if you ate asbestos rags, that would be a good way of getting a good dose on the larynx. Well, we've heard some evidence that may actually occur.

DR. MUSTARD: (cont'd.) And I wondered -- another problem as well, not only the pattern of exposure, but if the susceptibility to one effect of asbestos happened to be over a shorter time frame than effects normally might occur -- that other causes of deaths might occur, et cetera -- that you might -- there might, indeed, be an effect on the larynx in your data, but, because it's such a small effect, because the kind of exposure that might be influencing was only on a very small part of the work force, and because there are other competing causes of death, that it might all be masked in that information.

But I am curious, aside from those speculations -- have you any explanation, other than just pure chance, that rely -- it looks as though it's more frequent in 7(a) and 7(b)?

THE WITNESS: Well, what you're doing ---

DR. MUSTARD: It's simple, I know.

THE WITNESS: --- is comparing durations of exposure without taking any other factors into account, and it doesn't follow that they're comparable just on that account.

And, therefore, I mean -- I think that is not evidence, and I would, therefore, refer you to table 11 on page 20 -- sorry; table 10 on page 20 -- because, you see, what has been done here is to take those twenty-one deaths from laryngeal cancer and to take, for each one of them, three persons of the same sex, born in the same year, essentially, and then compare their distribution of exposures.

DR. MUSTARD: Assuming, since it's relative risk, that the other people subjected to low exposure didn't have an effect?

THE WITNESS: That is right.

DR. MUSTARD: And then my problem is, of course ---

THE WITNESS: That is perfectly right. I mean, then we have to go back to the SMR for laryngeal cancer -- and I

THE WITNESS: (cont'd.) can't remember what that is.

DR. MUSTARD: It's 1.5, from the low -- with the low exposure.

THE WITNESS: But we've only got seventeen, and I think we ought to look at the SMR, overall, for lung cancer -- or low exposure is -- I'm sorry; did you tell me the answer?

DR. MUSTARD: Yes; it's 1.45 for low exposure, 1.77 for medium, 1.19, and, for very high, I think it's 1.40. That just fascinated me, as I looked at that, and I realize it's sheer speculation.

THE WITNESS: Well, I mean, if you're looking at table 7(a), which is low exposure and short service, we have two cases of laryngeal cancer with an SMR of 1.45, but I'm sure you wouldn't really think that the 1.45 meant anything. You see, I mean, there are only two cases.

DR. MUSTARD: But you have, as you go across that whole column, you just have small cases.

THE WITNESS: Oh, I don't know; I mean, I -- I don't know any more than -- we don't -- I think it's true to say, isn't it ... I'd like to look again at the table which summarizes all our experience on laryngeal cancer.

Yeah; you see, on table 6, page 17, we have our laryngeal cancers spread out by length of service, and, as you say, there is some evidence that it is higher in the shorter ones; but, overall, the SMR, compared with Quebec, is sixteen cases observed and -- whatever it is -- it's an SMR of 1.07, which is on sixteen cases; obviously, the overall expectation of laryngeal cancer is very close to the Quebec average. So that -- well, there it is.

I mean, if you find that, overall, the cases are about the number you'd expect, and you find no evidence of a

THE WITNESS: (cont'd.) dose-response relationship, then I think you're forced to the conclusion that there's no good evidence in that series of it being due to it. But there's plenty of room for, sort of ---

DR. MUSTARD: Speculation.

THE WITNESS: --- speculation; yes.

DR. MUSTARD: Has anybody else ever observed any sort of increased SMR with comparatively short exposures, or is yours the only study where this ---

THE WITNESS: I don't know; you see, there aren't many studies on this. The Selikoff studies in insulation workers would presumably have some data on this, but, even there, you see, they didn't have very many laryngeal cancers. They had more than expected, but it was only something like seven observed and five -- four expected, or something. But there might have been more in their main series; I'm sorry, I don't know.

DR. MUSTARD: Well, let me change the subject. Looking at these tables -- and it's speculation again -- if you go to your 7(b), and I realized all these things from looking at this -- there are some interesting phenomenon, as you go through 7(a), (b), (c), and (d).

Pneumoconiosis as a cause of death doesn't become very big in those tables, until you get into length of service of twenty or more years; it begins to surface in the five-to-twenty-year table, but, all the way long, through the rest of it, there's respiratory tuberculosis, other respiratory disease, and heart disease.

But, as you move down in those tables, it certainly looks as if, as you've indicated, that the heart disease SMR starts to ---

THE WITNESS: Seems to come up; yes.

DR. MUSTARD: And then you see cerebrovascular disease, in 7(c) and 7(d), starts to come up, with the high -- in the very high doses.

You mentioned, in response to a question this afternoon, that that doesn't surprise you, and I suppose, I guess it shouldn't, in the sense that you think that's part of the vascular system response to pulmonary disease; but do you have any further thoughts on that subject?

THE WITNESS: Well, I suppose these are people who had strokes, mostly; and I would still sort of submit that, if you have a stroke and you've got some asbestosis, you're rather quicker to get the pneumonia that kills you than if you didn't; I don't know. That would be my reaction.

DR. MUSTARD: I see.

And then, finally, would it be reasonable to suspect that, over the time frame that these studies and documentation goes on, that among the subjects classified as pneumoconiosis, possibly from respiratory tuberculosis, and other respiratory, that there might be some overlap; that, indeed -- that you could almost, somehow or other, if you wanted to look at the full story on the lung, to save you looking at all those together -- and, if so, has anybody ever done that, just sort of lumped them together --- [coughing].

THE WITNESS: Well, one could simply add them together, of course. I think one of the things -- it is in this paper somewhere; it does draw attention to the fact that we have quite a marked era effect in tuberculosis throughout Canada.

But in the 19... -- the first part of the century, tuberculosis, in rural Quebec, was quite an important cause of death -- it was an important cause of death; so that, in fact, we do say somewhere that this analysis is, in fact, limited to

THE WITNESS: (cont'd.) deaths after 1950.

There were a disproportionate number of deaths from tuberculosis before 1950, and that was at a time when there was some concern about whether, you know, was there the silicosis all over again, was there evidence of tuberculosis and asbestosis interacting. And I don't think there was very much evidence found in those days.

I think -- we don't know; I mean, I think the answer is that it appears, as you rightly say, that there is a fairly definite dose effect with tuberculosis ---

DR. MUSTARD: On several of the ---

THE WITNESS: Yes, yes; I mean, that one, 7(d), it's quite marked. Now, the number of cases is not large, but it's quite marked. That could be misdiagnosis. I mean, I think this is one area where pulmonary tuberculosis could quite well be diagnosed rather than -- shall we say, if the man was known to have had tuberculosis, it could well be that he was attributed as having T.B., where perhaps it would be more true to say he had pneumoconiosis.

At the same time, if you have pneumoconiosis and you also have tuberculosis, they're a bad combination, even if they don't interact.

DR. DUPRE: Counsel, any further questions?

Well, Dr. McDonald, I think I can look with confidence on at least one score -- speak unanimously for your entire, occasionally rambunctious class. [Laughter.]

You have been most kind to be with us these last two days, and your performance, indeed, has been a pedagogical virtuoso. Thank you so very, very much, indeed.

We now rise, Miss Kahn, until 10:00 a.m. on Monday, the 29th of June; is that correct?

MISS KAHN: Right.

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